

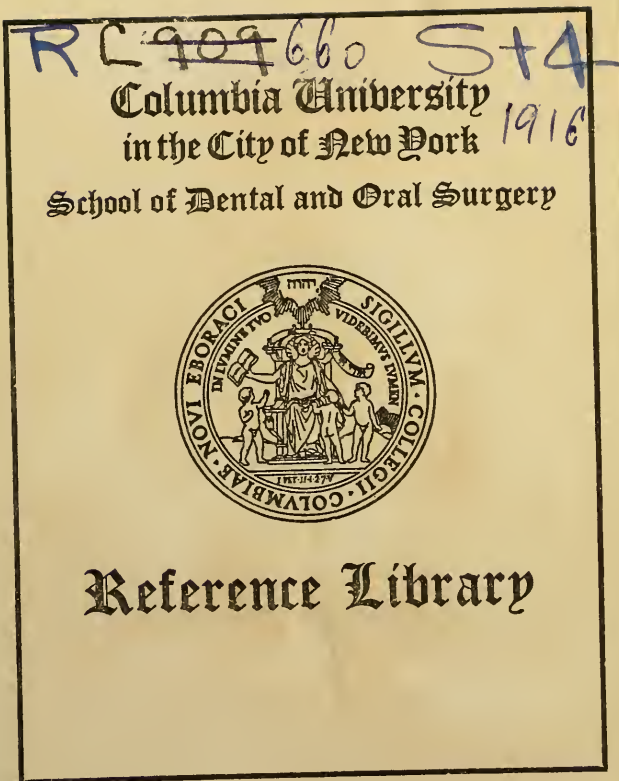
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FASTING AND UNDERNUTRITION IN THE TREATMENT OF DIABETES

BY

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PREFACE

Fasting (*not* starvation) has an assured place in diabetes therapy, a place, however, which is not by far as important as some modern enthusiasts are wont to make us believe. Fasting without being followed by undernutrition is entirely valueless so far as the management of diabetes is concerned.

In the preponderating majority of instances of diabetes a protracted fasting-undernutrition regimen is, fortunately, not indicated. In all such cases we do more lasting good without incurring any risks by following the older approved methods of dieting.

Only the advanced and severest forms of diabetes which are no longer or never were beneficially influenced by ordinary antidiabetic dieting are apt to be appreciably improved by continued fasting and undernutrition.

This little book is based entirely upon personal observations and experiences. It will be found that it differs, in certain essentials and in a number of details, from the rules and regulations laid

down by others in articles dealing with the same theme. The discussion of hypothetical subjects has been avoided in the main text (Part I). A few more or less theoretical points, particularly the question of the ketones, have been dwelled upon at greater length in Part II, the supplementary portion of the book.

HEINRICH STERN.

New York.

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PART I

FASTING AND UNDERNUTRITION IN THE TREATMENT OF DIABETES

I

INTRODUCTORY REMARKS

There is no pathological condition in the clinical control of which dietary treatment plays such a paramount influence as in diabetes mellitus. There is no other agent except a proper regulation of the diet that has ever been of any real value in reducing the two cardinal symptoms of diabetes: weakness and glycosuria. This fact has been definitely recognized ever since the advent of the physiological school of medicine.

Diabetes is and remains a wasting disease. By a rational system of dieting adapted to the individual case and to each period in the course of the affection, the painstaking clinician is often enabled to stem its progress—or rather its rapid progress—for the time being. This dietary individualization consists of the *periodical* diminution or the *temporary* withdrawal of one or all the types of nutriment.

The almost universal tonic for the confirmed

diabetic is the permanent, or almost permanent, complete withdrawal of all the sugar-containing aliments, and the greater or lesser temporary reduction of starchy foods. Upon this plan of withholding and decrease the management of the average case of diabetes has been based since the times of Pavy, Külz, Bouchardat, Ebstein and Naunyn.

There are numerous modifications of the sugar-free and starch-poor regimen. In the end all pertaining restrictions and modifications amount to the same thing: to render the patient stronger and more resistant, and sugar-free.

A logical outcome of Chittenden's "Physiological Economy in Nutrition," published in 1904, was that about two or three years later the proteins, especially those derived from meats, were also to a certain extent withheld from the diabetic. Finally a few clinicians decided that the reduction of all the types of nutriments was the *sine qua non* in the treatment of diabetes. While it was the accepted dictum before the investigations of Chittenden that the diabetic should be maintained in a rather continuous state of over-nutrition, a doctrine has lately been promulgated according to which the life of the diabetic should be one of everlasting renunciation and undernutrition. The first therapeutic conception is based on the assumption that diabetes is a wasting disease; the latter conception on the observation that a few diabetics get along better and become

more active after they have lost ten, twenty or more pounds.

On the face of it, however, the treatment of the average case of diabetes by a system of continuous undernutrition bears the stamp of inexperience, irrationality and harmfulness. Such a plan of treatment has its place in a small proportion of the cases of diabetes, in such cases in which besides a persistent glycosuria, a pronounced and obstinate ketonosis (acidosis) and rapid decline of body-weight and strength are present.

Here, it is true, fasting and undernutrition may produce wonders; in the preponderating majority of instances of diabetes, however, the patient will get along much better, and is apt to remain a useful member of society for a number of years if he be not kept below par.

On the other hand, forced feeding in diabetes is not always devoid of danger. Instead of assisting in the adjustment of the activity of the liver, continued overalimentation is bound to diminish and undermine, sooner or later, the function of this most important organ of the human body. Again, a liver that is not functioning perfectly can neither prevent the alimentary poisons from entering into the general blood stream, nor is it able to cause normal cleavage of the intermediary products of metabolism.

That total abstinence from food for from three to four successive days renders the urine of the

patient with severe diabetes sugar-free and "disintoxicates" the diabetic organism, was the important observation of G. Guelpa, of Paris. This clinical discovery has proved of great moment in the management of both (1) the grave types of diabetes, and (2) the overnourished cases of diabetes in which the liver no longer affords the necessary protection against the various forms of autointoxication. While it is true that the discoverer's own hypotheses respecting the rationale of the fasting treatment of diabetes are to the greater part scientifically untenable, it is nevertheless Guelpa who has shown us a way out of the bewildering vagaries of diabetes therapy. We should never forget that it was this French clinician who first in all the world declared that all food may be withheld for a number of days from a human diabetic without inciting ketonosis (acidosis) and subsequent coma. By one leap the treatment of severe diabetes has suddenly made more progress than it has for the previous quarter of a century.

Guelpa's "cure by privation" (abstinence from food and purgation) consists of the following:*

First. The taking every day for two, three or

* Guelpa: Starvation and Purgation in the Relief of Disease. Brit. Med. Ass., July, 1910. Published in Brit. Med. Jour., Oct. 8, 1910. Guelpa: Autointoxication and Disintoxication. Translated by F. S. Arnold, B.A., M.B., B.Ch. (Oxon.), New York, Rebman Company.

four days of a bottle of Hunyadi Janos water, warmed if possible, or from 40 to 55 c.c. (mils)=one and one-quarter to one and three-quarters ounces of castor oil, followed by about 750 c.c. (mils)=one pint and a half—of water.

Second. The abstinence during this period of all kinds of food.

Third. The free imbibing of water (not carbonated), weak tea without milk, toast and water, fruit infusion, etc. (Up to the time of the publication of the translation of his book, Guelpa had avoided any drug treatment whatever, as he wished to prevent any self-deception concerning the therapeutic influence of the treatment.)

Guelpa declares that it is only rarely that a patient has difficulty in completing his three or more days' fast. This he finds especially true if the purge and the other liquids that are imbibed during the fasting period are taken warm. The method of treatment usually calls forth pronounced improvement in the patient's state of health. In no instance had any aggravation of the disease resulted on account of the fasting and purging.

The benefits accruing from this plan of treatment are, according to Guelpa, the following:

First. Total disappearance of the annoying and distressing sensation of hunger.

Second. Marked decrease of the intestinal bacteria, and therefore a pretty perfect state of disinfection of the intestinal tract.

Third. Pronounced reduction of thirst.

Fourth. Suppression or conspicuous decline of perspiration, even in the heat of summer.

Fifth. Production of normal, periodic sleep, very refreshing though somewhat shortened in length. The patient, awakening passes through no stage of drowsiness; full mental activity ensuing immediately.

Sixth. Firmness of the pulse and diminution of blood pressure, and increase in hemoglobin, red cells and leukocytes.

Seventh. A decrease in the volume of certain visceral organs, particularly the heart and liver, with greater and easier lung expansion.

Eighth. Continuous loss in body weight, at the average rate of two pounds a day. Concurring therewith the activity of the heart and other organs becomes less oppressed.

Ninth. Disappearance of joint and muscular pains and the production of a feeling of agility, liveliness and well-being.

Excepting a few loose statements concerning a low protein intake, Guelpa has not devoted much attention to the *dietary treatment following the fasting periods*. More or less he has concentrated his therapeutic endeavors upon that what is now understood as the "initial fast," and has given no or but scant rules how to proceed as regards the food-intake after the fasting period is broken. Yet the question how to go ahead after the fasting days without occasioning the reoccurrence of

sugar and ketones is the only intricate one connected with the plan of treatment. The fasting period itself offers no difficulty whatever.

Fasting and undernutrition in the treatment of diabetes must never assume a stereotyped character. Some patients do well on a one or two days' fast and a week of undernutrition; in others the fasting period should extend over five, six or more days, and the subsequent undernutrition should be continued for one, two, three or more months. In some diabetics of the graver types a fast day weekly or fortnightly and a slight reduction of the diet may keep the disease in check, in others the duration of the periodical fasting term and a much more decided reduction of alimentation are necessary to accomplish the desired end.

Fasting with subsequent undernutrition must go hand in hand in the treatment of the severe forms of diabetes. This fact was already recognized in some degree by Naunyn when he advocated the interpolation of "green days" in the dietary of the patient affected with grave or advanced diabetes. These "green days," however, are to all intents and purposes fast days, as the patient obtains on them but a certain amount of green vegetables whose low starch content is not readily attacked by the digestive juices.

Undernutrition in diabetes, as I understand and uphold it, is not the reduction of the intake of one class of foodstuffs, but of all of them. The

proportion in the diminution of the various articles of food must, however, of necessity differ in the various phases after the fast. Fats, particularly those of high melting point, may practically always be permitted, though they may on rare occasions give rise to a slight ketonosis (acidosis) or aggravate an already existing one. Proteins of animal origin, in more or less reduced amounts, must be resorted to soon after the fast. The patient simply cannot get along without them, despite the assertions of some clinicians to the contrary. Great care, however, must be exercised that the ingested proteins do not overburden the liver. The starches should be added latest and then only in the form of vegetables containing at first not more than five per cent. of carbohydrate matter which is not readily elaborated by intestinal activity.

It is now almost six years since I started to make use of Guelpa's method in suitable cases. Naturally, I have modified and amplified it according to the needs of the manifold cases which have come under my observation since that time. The general plan of treatment by fasting and undernutrition, as evolved by me, differs in more than one respect from similar endeavors. This plan of treatment which is minutely dwelled upon in the following pages is designed as a guide, pure and simple, and as such it merely points to a course of procedure, but it is readily capable of modification and adaptation to individual demands.

II

TECHNIC OF THERAPEUTIC FASTING IN DIABETES

Where to take the fasts

While the hospital, on account of its laboratory and other facilities, may be the best place in which to let the patient take his initial fast, the home, for reasons presently to be explained, is after all to be preferred when the patient has to submit to frequently repeated fasting periods. The fasts in order to do any good should be undertaken at certain, preferably regular intervals. One fast is of little consequence, and it should be remembered that one swallow does not make a summer.

The arguments in favor of home treatment are, (1) the simplicity of the management of the case itself; (2) the simplicity of the necessary tests which, if need be, may be performed at the bedside by the physician, a nurse or by the patient himself; (3) the accustomed atmosphere which is much less apt to depress the patient than the hospital milieu, and (4) the inexpensiveness as compared with the extravagant hospital charges.

The patient should be resting in bed during the entire course of the fast. The room should have a southern exposure, if possible, but it must

not be artificially heated. The toilet, if conveniently located, may be used by the patient. If this be not the case a commode must be employed.

The attending physician

During the inaugural fasting period the attending physician should make daily calls and personally assure himself of the patient's physical condition. He should perform or should have performed a blood examination for glucose before the fast is started. In the beginning it is well to examine for blood-sugar every third or fourth day. This, however, is not imperative. The urine should be tested every day for grape-sugar and the ketone substances, especially acetone and diacetic acid. (The examination for beta-oxybutyric acid is difficult and time-consuming, and is by no means essential in the general run of cases.)

During subsequent fasts—provided no complications ensue—the physician need not pay any calls. He should, however, make a daily urinary assay. If there be any indication, an examination for blood-sugar should be made at the beginning and conclusion of each subsequent fast. The physician should give his orders either to a nurse, a member of the family or to the patient himself.

The nurse

The initial fasting period should be under the direct and continued supervision of a nurse. The

nurse, as a matter of course, receives her instructions from the attending physician to whom he or she should report the patient's condition as often as the exigencies of the case demand. It is the nurse who has to minister to the wants of the patient and who has to keep him as comfortable as possible. It is the nurse—and this should be particularly the case during the initial fast—who forms besides the physician the sole means of communication between the patient and the outside world.

The nurse should keep a chart of the temperature, pulse and respiration which should be recorded from three to five times during the twenty-four hours. The temperature should always be taken in the rectum unless a disease of the same precludes this. The condition of the skin should be recorded, whether it be warm or cold, dry or moist, etc. Besides an exact account, both as to the amount and the time of the intake of liquids and the output of urine and feces must be kept by the nurse. In subsequent fasts, when the diabetic state of the patient is no longer an unknown factor to the medical observer, it is not essential that a detailed chart be kept in the average case.

The nurse need not belong to the privileged "registered" class. Any member of the family with a little common sense, possessing some tact and devotion, is able to nurse an uncomplicated case of the graver forms of diabetes during all but the initial fasts. Many servants who watch

the doings of the trained attendant during the initial fasting period will make excellent nurses for cases during the periods of fasting and undernutrition. Indeed, my best nurses for patients suffering from nutritive disturbances I have recruited from the ranks of the "unregistered," the common garden and field variety of attendants. Last but not least, they receive only from one-third to one-half the salary which the "R. N." exacts.

The patient his own nurse

I am often asked by the patient why he cannot act as his own nurse during the therapeutic fasts. Excepting, of course, the initial fast when valuable data for the future guidance of his case are to be gained and when he is necessarily to be kept under close medical surveillance, the question is certainly justified as, roughly speaking, about half of the cases get along quite nicely without any special assistance. It is my experience that male diabetics may be more trusted in this respect than their female fellow-sufferers. As a rule, the members of the stronger sex have a much better conception of their disease than those of the weaker one, and men have a much keener appreciation than women of the necessity that something be done for their infirmity. Of course, when entirely without the help of somebody the diabetic patient cannot keep his fast. Someone has to make his bed, prepare the per-

mitted liquids for him, and do other chores. However, as just stated, about one-half of the cases need no *special* attendance whatever.

The patient himself

It goes without saying that the patient must have full confidence in his physician. He should talk matters over with him, and not start fasting unless he feels assured that this therapeutic measure is going to benefit his condition. The patient should become acquainted with the fact that the fast is to be immediately followed by a more or less protracted period of undernutrition. He must foster a certain degree of self-denial and assume a mental attitude of submission, good will and trust. He must be entirely free from mental unrest, and for this reason should put his house in order before he attempts the fasts. He must not question the orders of his physician once the fast has begun. Worry, exaltation and excitement are only too apt to aggravate a case of diabetes and to increase the intensity of the sugar excretion.

The patient will note that when the ice is once broken, viz., after he has undergone the initial fast, that there are worse things than abstaining from food for a number of days. In subsequent fasts many patients are masters of the situation, indeed so much so that everything connected with the therapeutic fasts, even the slightest detail, may be left to their own discretion and execution.

The cooperation of such patients with their physicians spells success—if a favorable issue can be obtained by this plan of treatment.

Duration of the fast

The average ketonemic diabetic voids a sugar and acetone-free urine in from three to five days after starting the initial fast. A majority of these patients cease to excrete sugar within forty-eight or sixty hours. In some patients the glycosuric symptom already terminates after the omission of two or three meals; in others it persists for six days or even longer. Again, there are cases of the severest type of diabetes which no amount of fasting will render sugar-free. Generally speaking, the initial fast should not come to an end when sugar ceases to be excreted, but should be continued for another twenty-four or forty-eight hours.

It always takes longer to render the patient's urine free from ketones (diacetic acid, acetone, beta-oxybutyric acid, etc.) than from sugar. In a rather considerable number of instances—twenty-five per cent. approximately—no amount of fasting will accomplish this feat. In these cases prolonged starvation treatment is out of place; the accustomed diet should be taken up as soon as possible.

Subsequent fasts need hardly ever be protracted unless the intervening period has been an exceptionally long one. The intercalation of a

hunger-day, as advocated by former authors, will often suffice.

Frequency of the fast

No iron-clad rules concerning the frequency of the therapeutic fasts can be propounded. Once the inaugural fast is over and the severity of the case determined (*i.e.*, the readiness of the disappearance and reappearance of the urinary sugar and ketones), it is a simple matter to decide when and how often subsequent fasts should be undertaken. The decision resolves itself in the answering of the following two questions: *Firstly*, has the inaugural fast been of benefit to the patient; *secondly*, is the patient to be kept entirely sugar- and acetone-free during the interval.

It should be the physician's endeavor not only to prolong life, but also to prolong the usefulness of his patient. For years I have advocated that the diabetic with low carbohydrate tolerance and a tendency to acetonuria should make a fast-day of that which is euphoniously called "the day of rest." If possible, I let him also add the Saturday half-holiday. By cutting out the Saturday mid-day meal and permitting a very moderate supper on Sunday, a fasting period of about thirty hours is obtained without interfering with the patient's business. I am in the habit of allowing a small amount of food on Sunday evening in order that the patient is able to resume work the next morning, which he does after taking his

regular breakfast. These weekly fasts are a great boon to the average patient affected with a severe type of diabetes, and often enable him together with the restricted diet during the week to keep above water in a physical, financial and social sense.

In many cases a one-day fast every second or third week may suffice, in others of the severer and severest types more protracted fasting periods—for from two to four days—should be instituted every two weeks. Thus it may be necessary that four days out of every fourteen must be spent fasting. It should not be forgotten, however, that a certain proportion of these cases are not only not benefited but actually harmed by this treatment. The initial fast enables the clinician to recognize the cases not suited for the fasting-undernutrition treatment.

The indications for instituting protracted fasting periods other than the initial one, are: progressive weakness of the patient, certain complications like gangrene and infection, very low or negative carbohydrate tolerance, pronounced ketonosis (acetonuria, acidosis, etc.), heart disease (fat heart), marked obesity and disease of the liver (hepatic inefficiency) and kidneys (renal insufficiency).

All subsequent protracted fasting must occur while the patient is resting in bed and under the same general conditions as the inaugural fast. A single weekly fast-day, however, does not call for

bed-rest, especially not in summer time. The patient may spend the day in the open air, reclining in a steamer chair or other comfortable piece of furniture, but away from the hustle and bustle of every-day life.

Other essentials in therapeutic fasting

A. *Purging*.—Inasmuch as fasting in the graver forms of diabetes is considered by Guelpa to be a method of disintoxication, he combines the treatment for the sake of greater efficacy with purgation. The natural Hungarian bitter waters, Carlsbad water and the less concentrated solutions of sodium or magnesium sulphate are best suited for the purpose provided there be no kidney disease.

Of the natural mineral waters not less than two tumblerfuls, heated to about 130 deg. F., should be taken at one time, preferably in the morning. If the bowels have moved less than twice during the day, the dose should be repeated in the late afternoon. If a solution of sodium or magnesium sulphate should be prepared extemporaneously not more than one and one-half teaspoonful should be added to each glass of warm water. Two glasses of the solutions are a dose. The purge is to be taken in the morning and, if necessary, also in the early evening. In instances with impaired kidney efficiency castor oil should be substituted for the solutions of Epsom and Glauber's salts. It should be administered once

a day in doses from 30 to 60 c.c. (mils)=1 to 2 ounces. Care must be taken that it be not admixed with any sweetening material.

In the average case these purgatives must be exhibited every day as long as the fast lasts. They should not be replaced by any other laxatives, especially not by drugs containing sugar, such as elixirs of cascara sagrada, magnesium citrate, compound powder of licorice, etc.

B. *Drinking*.—The continuance of the normal physico-chemical processes in the organism demands that a certain amount of liquids be imbibed. At the same time fluids are the carriers of a great number of excretory substances. Water may practically be partaken of at pleasure by the fasting diabetic provided there is no contraindication offered by advanced cardio-vascular disease and a general hydremic condition. The patient may drink from 20 to 40 c.c. of water for each kilogram (from a little more than $\frac{1}{2}$ ounce to somewhat more than 1 ounce for each pound) of body weight in the twenty-four hours. A man weighing 60 kilograms (132 pounds) may therefore take daily from about 1200 to 2400 c.c. (35 to 50 ounces) of water. Only still water should be permitted, as carbonated waters are apt to produce gastro-intestinal and vesical discomfort.

Some of the necessary liquid may be given in the form of a weak infusion of tea. Of course, this must be taken without milk or sugar, but

some lemon juice is permissible. Three, four or even five cups of weak tea may be drunk during the twenty-four hours, but the liquid thus imbibed should be deducted from the total amount that is allowed. The tea (as well as other fluids) may be taken as warm as desired unless there be a gastric or duodenal disease, ulcer for instance. Then, of necessity, the liquids must be taken lukewarm or cold. Occasionally tea is not well-borne; it may occasion headache or a slight gastric disturbance, an undue diuresis, or it may agitate the patient, preventing sleep. In such cases I am in the habit of ordering a very weak infusion of chamomile which is a wholesome and satisfactory drink. In rare cases I permit coffee. However, it usually overstimulates the patient and the reaction never fails to make itself known. This reaction manifests itself by a pathological depression. Not more than two cups of rather weak coffee should be allowed for each day of the fast.

If the fast is to be continued for longer than two days some hot beef broth, a cupful at midday and the same amount five hours later, will act as a mild stimulant. The quantity of the broth should also be deducted from the total amount of permitted liquid.

Sparers of body tissue

There are certain substances the ingestion of which may prevent too rapid and too pronounced a decline of body weight when the organism is in

a state of therapeutic fasting. The best known substances of this class are alcohol and gelatine.

A. *Alcohol*.—Alcohol is by no means as valuable a saver of body structures as gelatine. Positive proof that it saves the tissues in healthy man is absolutely wanting. However, alcohol is a time-honored adjuvant in the treatment of a number of wasting diseases, and its stimulating action is certainly of benefit to many patients. Small doses of alcohol, that is about 0.5 c.c. per day for each kilogram of body weight (about one drachm per pound) may be permitted. A person weighing sixty kilograms (132 pounds) could therefore consume in the neighborhood of 30 c.c. (one ounce) of alcohol per day. As the distilled liquors contain on the average about fifty per cent. of alcohol, a patient of above weight may take 60 c.c. (two ounces) of whiskey or brandy in the twenty-four hours. In exceptional cases twice the amount even may be permitted. These alcoholic beverages should be well-diluted with plain water, and given in small doses every two, three or four hours.

B. *Gelatine*.—Gelatine does not appear to be a builder of body-albumin, but it spares circulatory proteid. Hence, it prevents or limits the loss of body-albumin. The albumin-saving property of gelatine is at least twice as large as that of the fats and carbohydrates, as 100 grams of it can replace 36 grams albumin, that is about 175 grams of meat. Of course, the gelatinous substances can-

not entirely prevent loss of tissue albumin, and, for this reason, a certain amount of albumins proper must ordinarily form a constituent of the nourishment. In the gravest forms of diabetes when glucose and ketones are being excreted in considerable quantities, the ingested gelatine still produces bodily energy, and averts rapid body waste without increasing the sugar output in any marked degree.

Gelatine may also be a sparer of body-fat. However, its influence in this respect is more limited than in regard to the body-protein. Still, 100 grams gelatin prevent disintegration of 25 grams body-fat, a fact which may be made use of in the suppression of the ketone substances (acetone bodies). In the latter respect it is, indeed, a much more valuable substance than alcohol.

The tissue-sparing properties of gelatine are only limited by the comparatively small amount of the substance which the organism will tolerate without aversion.

It is always best to have on hand two or more differently flavored gelatines when treating a diabetic. Gelatines to which is added some lemon juice or some beef extract, beef juice and their like are especially cherished by the fasting patient. Such preparations may be given alternately, from a teaspoonful to a tablespoonful at a time. The diurnal intake should not exceed 10 or 15 grams (one-third to one-half ounce) of gelatine (in the raw).

Drugs

Medicines have little or no influence upon the excretion of sugar or the ketones and are certainly out of place during the therapeutic fast of the diabetic. The much vaunted, but in the suppression of acidosis entirely useless sodium bicarbonate, is included in this pronunciamiento.

Even if there were any proof of the antiglycemic or antiketonic properties of certain drugs, their employ would still be contraindicated in the fasting diabetic for the reason that the therapeutic influence of the fast alone be recognized. This is especially true in the inaugural fast.

Again, the fact must not be lost sight of that in the fasting person most medicines exert their physiological effects much more readily than in the individual that is properly nourished. If, therefore, the attending physician, for one or the other reason, does not think he is able to get along without a certain drug, he should, if it be a potent one, prescribe it in smaller than the accustomed doses. Under all circumstances, however, he should rid himself of the superstition that any medicine which he may order is able to control or modify the sugar or acetone output of his patient.

The average diabetic on a fast sleeps on and off from twelve to eighteen hours per day. So long as his mental condition does not prevent it he falls asleep for similar reasons as the hibernating

animal does. Should a hypnotic be necessary, morphine in doses from 0.0075 to 0.005 gram ($\frac{1}{12}$ to $\frac{1}{8}$ gr.) is not alone the safest and promptest, but withal the most valuable of this class of remedies, as it retards the processes of general metabolism, thereby acting as a tissue sparer.

Medication for local pathological states, as gangrene and infection, is, of course, necessary. A remedy thus employed differs entirely from a drug that is supposed to ameliorate the diabetic condition as such.

III

BREAKING OF THE FAST AND UNDERNUTRITION
IN DIABETES

While the technic of therapeutic fasting in diabetes is a rather simple matter, this is by no means the case with the breaking of the fast, for this entails a variety of precautions which I may be allowed to embrace in the principle "Individualization" of the patient.

Individualization of the patient

There are few measures demanding a stricter individualization of the patient than the re-starting of eating without occasioning sugar and acetone after a therapeutic fast of the diabetic. It is not sufficient to render the patient sugar-free or, in favorable cases, free from acetone substances. This freedom from sugar, and of ketones, should be maintained for as long a period as possible, provided the patient becomes not unduly emaciated and feeble and a burden to himself and his family. The moment the patient rapidly fails—sugar or no sugar—ketones or no ketones—, he has to be supported by a diet that does not keep him in a state of undernutrition. It is surprising to note how some diabetics thrive

on a diet that is none too strict soon after a therapeutic fast. Of course, the sugar may and most always does return, but the patient may increase in body weight, strength and endurance.

The great majority of the diabetic patients, however, are not benefited at all by the fast unless this be immediately followed by a more or less prolonged state of undernutrition; and furthermore, there is a small group of diabetics which is actually worse off after than before the fast. This is the case when the sugar and acetone excretions become quickly re-established on account of a wrong diet.

Fasting therapy is indicated in about twenty per cent. of all the cases of diabetes. These cases recruit themselves from among the severer types, as a rule. In these cases the course of the diabetic affection has either been a rapid one right from the start or the patient has had the disease for a long period. In either eventuality he has lost a good deal in weight, and his bodily force, efficiency and resistance have markedly diminished. In patients of this class the ordinary methods of antidiabetic dieting usually no longer avail much, and the fasting treatment must and should be given a fair trial. It stands to reason, however, that the management of these cases must be strictly individualized. Neither should the fast cause a too rapid or too great a decline of body weight nor should it cause diminution of strength beyond reasonable limits.

Some medical enthusiasts have declared that the loss of weight *per se* exerts a salutary influence upon the diabetic constitution and increases tolerance by allowing the diminished function of the pancreas to recuperate. Such an assumption can only have been dictated by purely academic experiments and clinical inexperience. In the grave forms of diabetes in which a system of fasting periods is to be instituted, the motto, "To have and to hold" must be paramount, and be adhered to as closely as possible.

On the other hand in the general run of cases of diabetes, furnishing approximately eighty per cent. of the affection, fasting treatment, though not invariably contraindicated, seems out of place as a measure of routine. To this great category belongs the so-called diabetes of the obese, usually one of the mildest types of diabetes. Dieting or no dieting, obese diabetics lose from ten to thirty pounds on the average during the first year of the affection. If they are subjected to an anti-diabetic regimen (which in sum and substance is identical with an antiobesity diet) they decline in body weight on account of the ingesta, changed quantitatively and qualitatively. If on the contrary they continue to subsist on their habitual food, they lose just the same because the diabetic deterioration is not being curbed and counter-worked. Diabetics of this class unquestionably fare better if they lose some weight; this, however, will ensue at all events.

As already stated, four-fifths of all diabetics need not ordinarily submit to any fasting periods. To be sure, fasting may not necessarily do any harm in these cases, but a strenuous treatment of this sort is comparable to shooting at sparrows with cannon balls.

The diet after the inaugural fast—undernutrition

First and second days.—When the urine has been sugar-free for about two days the tea, beef broth and alcohol, which were allowed during the fasting period, should be continued, the gelatine preparations may be withdrawn, and from three to four yolks of eggs are to be added during the subsequent twenty-four hours. The yolks are best given by incorporating them with the permitted liquids. One-half of one yolk may be mixed with the tea at breakfast time, another half with the whiskey around ten or eleven o'clock in the morning, a whole yolk with the broth at the luncheon hour; half a yolk with tea at about four P.M., and a half or whole yolk with whiskey in the evening. (Concerning the rationale of the yolk ingestion in the graver forms of diabetes see page 111.)

If a mixture of the urine voided between six P.M. of that day and seven o'clock the following morning is still free from sugar, the amount of broth and yolks may be doubled while that of tea and alcohol should remain unchanged. If the urine derived from the period between six P.M.

and seven A.M. does not show clinically demonstrable amounts of sugar, the following dietary for the third and fourth days after breaking the fast may be ordered.

Third and fourth days.—

Breakfast: Cup of tea or coffee, without milk or sugar.

Two soft-boiled eggs with two extra yolks.

10.30 A.M.: Egg-nog (20 c.c. ($\frac{2}{3}$ ounce) whiskey, 75 c.c. ($2\frac{1}{2}$ ounces) water, one egg yolk).

Midday: One or two plates of beef broth (prepared with green vegetables, such as parsley, leak or onion, celery tops, strained) with one yolk per plate stirred in.

Spinach, dessert-plateful (75 grams= $2\frac{1}{2}$ ounces) with one whole egg.

4 P.M.: Whiskey (20 c.c.= $\frac{2}{3}$ ounce) with one yolk.

Supper: One plate of beef broth (as above), with one yolk stirred in.

String beans, dessert-plateful (75 grams= $2\frac{1}{2}$ ounces).

Lettuce with French dressing and one or two yolks.

It is not necessary that the urine of the third day after breaking the fast be examined. The urine of the fourth day, which is to be accumulated for the same period six P.M. to seven A.M.

(the fifth day) will tell the story much better. If it contains no sugar, the following diet for the fifth day may be proceeded with.

Fifth day.—

Breakfast: Cup of tea or coffee, without milk or sugar.

Two soft boiled eggs with two extra yolks.

Two slices of bacon.

10.30 A.M.: Egg-nog as on previous day.

Midday: One or two plates of beef broth (prepared as on previous days), with one yolk stirred in for each plate.

Spinach, soup plateful (125 grams=4 ounces) with two whole eggs.

Four oil sardines.

String bean salad (French dressing).

4 P.M.: Whiskey (20 c.c.= $\frac{2}{3}$ ounce) with one yolk.

Supper: One plate of chicken broth, with three tablespoonfuls of cut giblets.

Brussels sprouts or kohlrabi, seasoned, but without butter or flour, dessert-plateful (75 grams= $2\frac{1}{2}$ ounces).

Salad of canned asparagus (eight to twelve whole asparagus).

American cheese (30 grams=one ounce).

If after this diet there occurs no urinary sugar, a larger amount of protein in the form of meat may be added to the food of the sixth day.

Sixth day.—

Breakfast: Cup of tea or coffee, without milk or sugar.

Ham (or bacon) and eggs (two eggs, 30 grams = one ounce ham or bacon).

10.30 A.M.: Egg-nog (as on previous days).

Midday: One or two plates of green vegetable soup (parsley, celery, onion, cabbage, cauliflower).

Breast of chicken (40 grams = $1\frac{1}{3}$ ounce).

String beans, dessert-plateful.

Lettuce or watercress, French dressing.

4 P.M.: Whiskey (20 c.c. = $\frac{2}{3}$ ounce), or tea, with one yolk.

Supper: One or two plates of green vegetable soup (as above).

Cabbage, dessert-plateful.

Dandelions or field salad, dessert-plateful.

American cheese (30 grams = one ounce).

If the urine continues to be free from sugar, the following sample of a diet may serve for the seventh day of undernutrition following the fast.

Seventh day.—

Breakfast: Cup of tea or coffee, without milk or sugar.

Ham (or bacon) and eggs (two eggs, 40 grams = $1\frac{1}{3}$ ounce ham or bacon).

10.30 A.M.: Egg-nog (as on previous days).

Midday: One plate of chicken broth with half tablespoonful of rice (cooked); some chicken giblets.

One lamb chop, or a slice of any meat (50 grams= $1\frac{2}{3}$ ounce).

Spinach, string beans, or Brussels sprouts (one even soup-plateful).

Lettuce, French dressing, as much as desired.

4 P.M.: Whiskey (20 c.c.), or tea, with one yolk.

Supper: One plate of chicken broth, without rice, but with some chicken giblets (three tablespoonfuls).

Any green vegetable (one even soup-plateful). Romaine or endive, French dressing, as much as desired.

Brazil nuts (30 grams=one ounce).

After a week, then, the patient obtains a daily ration yielding between 1000 and 1100 of available calories. This is approximately one-third less than he should receive at the end of the following week, and about half of the caloric food value he should be allowed at the end of the third week after the fast, provided his urine be still free from sugar.

During the period of undernutrition, that is, so long as the diabetic patient ingests food less than will yield about thirty-five calories per kilogram of body weight, he cannot, of course, pursue his usual occupation. It is not quite necessary

that he be resting all the time, but he should take only very moderate exercise and be in bed for not less than from fourteen to eighteen out of the twenty-four hours.

Second week of undernutrition

The bulk of the increase in calories during the second week after the inaugural fast should derive from the fats of high melting point. For reasons set forth elsewhere (pages 111, 133) the yolk of the hen's egg contains the fatty substances best suited for this purpose. For the very same reasons cream and butter should be withheld strictly. The daily ingestion of from ten to fifteen yolks, properly incorporated with the other food, almost every diabetic will submit to for a few weeks. This number of yolks yields approximately from 500 to 750 calories. The addition of but even ten yolks to the diet of the seventh day (see before) would almost make up for the daily caloric deficit during the second week. However, an increased amount of green vegetables and some more protein (egg-white, meat, fish) may be allowed to help in overcoming the caloric inadequacy.

In this connection I wish to state that the determination of the fat-tolerance is hardly necessary as far as the production of urinary sugar is concerned. The fat-tolerance should be ascertained because the low fatty acids may be possible progenitors of the ketone substances (acetone

bodies). In many hundreds of cases of diabetes in which I have employed the "yolk cure," I have seen a heightened intensity degree of the glycosuric symptom in less than half a dozen instances, while an increased ketonuria ensued in not more than three or four per cent. of the patients. An ordinary yolk contains over five grams of fat. Ten yolks embody therefore somewhat more than fifty grams of fatty material. This is less than one gram per kilogram of body weight of the average patient. As a matter of fact, the general case of diabetes undergoing the undernutrition treatment may have in the second week after the fast as much as two or three grams of fat of high melting point per kilogram of body weight.

Method of adding food

The food-increase should occur slowly and in the manner that not more than one and a half calories per day and kilogram of body weight be added. A man weighing 60 kilograms (132 pounds) should therefore receive a daily addition of ninety calories from day to day. This means more than six hundred additional calories on the last day of the week. Together with the food of the seventh day of undernutrition (after the fast) when the number of calories available from the food amounted to from one thousand to eleven hundred, this patient hence partakes at the end of the second week (after the fast) of an amount of nutriment almost maintaining him in a state

of metabolic equilibrium. His decline in body weight should not surpass one to two kilograms (two to four pounds) during the second week of undernutrition.

Here follows a sample dietary for the first three days of the second week (eighth, ninth and tenth days) after the fasting period. The figures in brackets are for the ninth and tenth days, respectively.

First three days of second week

Breakfast: Cup of tea or coffee, without milk, cream or sugar.

Bacon and eggs (3 eggs), 1st day 50 grams, 2nd day 60, 3rd day 70 grams bacon.

10.30 A.M.: Egg-nog (whiskey 20 c.c., two yolks).

Midday: One plate (2nd and 3rd days 2 plates) vegetable soup (parsley, celery, onion, cabbage, cauliflower, tomatoes, unstrained if desired).

Fish, mackerel or shad, boiled or broiled, egg sauce, 30 grams (40, 50 grams 2nd and 3rd days resp.).

Beef in any form, one small slice, medium fat, 70 grams.

Cauliflower, 60 grams (70, 80 grams 2nd and 3rd days resp.).

String beans, 60 grams (70, 80 grams 2nd and 3rd days resp.).

Cold slaw, French dressing, as much as desired.

4 P.M.: Whiskey, 20 c.c., or tea, with one yolk (2 yolks 2nd and 3rd days resp.).

Supper: Four oysters (5, 6 oysters 2nd and 3rd days resp.).

One plate of beef or mutton broth.

Omelette, two eggs.

Spinach, 100 grams.

Any green salad, French dressing, as much as desired.

The available calories of this diet vary from about 1150 on the eighth to about 1325 on the tenth day. By the end of the second week the patient should take approximately the following dietary in case the urine has remained free from sugar.

End of second week after fast

Breakfast: Cup of tea or coffee, with one yolk, but without milk, cream or sugar.

Bacon and eggs (3 eggs, 80 grams bacon).

10.30 A.M.: Egg-gelatine (one whole egg, one extra yolk).

Midday: One plate of okra soup (some green vegetables, tomatoes, teaspoonful of oatmeal).

Salmon, smoked, 40 grams.

Bean salad, 150 grams.

Beef in any form, medium fat, 100 grams.

Spinach, 100 grams.

Egg-salad (2 eggs), lettuce, French dressing.

Brazil nuts (30 grams without shells).

4 P.M.: Coffee-gelatine (half teaspoonful powdered gelatine, two tablespoonfuls water, three quarter's cup boiling coffee).

Supper: Anchovies or sardines in oil (3).

Chicken broth with half a tablespoonful of fine noodles.

Asparagus omelet (three eggs, eight stalks asparagus).

Cauliflower, 100 grams, with yolk sauce (two yolks).

Ripe olives (40 grams).

The foodstuffs in the last diet list yield no less than from 1750 to 1800 calories, an amount on which a patient weighing sixty kilograms (132 pounds) can very well get along for some time if he be at rest the greater part of the twenty-four hours.

Third week after the fast

By the end of the third week after the fast, the patient—in successful cases—will be able to ingest food yielding about 40 calories per day and kilogram of body weight without giving rise to either glycosuria or ketonuria. At the same time he should not have lost more than from six to eight kilograms (13 to 17½ pounds) in weight, and be rather more vigorous (resistant) than before he started to fast. If this be not the case the method of treatment here outlined is a failure in this particular instance. Recalling to mind that only in the severest forms of diabetes the

fasting-undernutrition method should find employment, it will at once be seen that the difference between 1800 calories at the end of the second, and 2400 calories at the end of the third week, cannot be made up by carbohydrates alone. Moreover, experience has shown that the ingestion of vegetables containing five or ten per cent. carbohydrate in amounts larger than 400 grams, is practically useless for the human economy inasmuch as the intestinal juices in a severe case of diabetes are insufficient to elaborate the starchy material from the mass of cellulose textures. The additional carbohydrate must, therefore, be derived from vegetables containing a higher percentage of starchy material. At the same time it should be easier of elaboration.

The tolerance for carbohydrate as such in the severer forms of diabetes is always below fifty grams per day; often it is nil. These fifty grams carbohydrate yield, at the best, 200 calories. Vegetables with a low carbohydrate content (five per cent. or less), for reasons just mentioned, hardly furnish any starchy material to the diabetic organism. They fill the gastro-intestinal tract, giving thereby a certain feeling of satiation; they also supply a certain amount of various salts, but they do not contribute to any extent to the combustion processes within the body. In planning the food intake up to the end of the third week after fasting, it will be found that, as far as their nutritive-calorific value is con-

cerned, the green vegetables by themselves have not been taken by me into any account whatever. They are solely added to the dietaries in order to meet physical and mental satisfaction.

Favorable cases, those which have hitherto remained sugar and ketone-free, should receive about ten grams more fatty material (no cream or cheese) and the same amount of protein every other day during the ensuing week. Furthermore, these patients should be tested for their carbohydrate tolerance. For this purpose I prefer the small, hard potato (new, if possible) which contains approximately twenty per cent. starch. I order the potatoes in any of the following forms: Boiled in their jackets; French fried, or Saratoga chips.

Determination of carbohydrate tolerance

The patient receives the diet of the end of the second week after the fast (or its equivalent) and the fat and protein addition just pointed out. To this is added five grains potato in the form of Saratoga chips at the midday meal. If the urine for the ten hours following the potato intake be sugar-free, five grams of the Saratoga chips may be again added to the midday meal of the following day; in addition five grams of the same may also be incorporated with the supper of this day.

In case the urine voided between 1 P.M. and 7 A.M. the next day is sugar-free, seven one-half

grams of the chips should be added to the mid-day repast and the same amount to the supper. If the afternoon-evening-night urine shows no sugar, the patient may have twenty grams chips, ten grams respectively for the midday and evening meals. If sugar cannot be demonstrated in the urine twenty-five grams chips, half at noon and half in the evening, should be allowed on the following day.

In case the urine continues to be sugar-free the twenty-five grams of potato chips should be continued for a week or ten days as part of the daily ration of the patient. After this period the tolerance determination for carbohydrate may be continued, but in the manner that only very small amounts of the carbohydrate, say two or three grams, be added every day or, what is still better, every other day. However, it will be found that but few of the severe cases of diabetes will tolerate more than thirty or thirty-five grams available carbohydrate from day to day.

In exceptional cases the carbohydrate tolerance increases for a certain period or from time to time. With the essentially identical intake of food the carbohydrate tolerance becomes suddenly greater so that no glycosuria supervenes after thirty or even forty grams of carbohydrate, whereas on the previous day fifteen and more grams of glucose were excreted with the urine. This phenomenon generally disappears as rapidly as it has ensued and, in my opinion, has

nothing to do with sparing of the function that presides over the carbohydrate metabolism.

Subsequent diet

The subsequent diet-question in severe cases of diabetes resolves itself in particular in the decrease or occasional re-increase of the carbohydrate intake and the periodical interspersing of fast days. In this respect the dietary management of severe cases of diabetes following an inaugural fast and the graded system of undernutrition does not differ essentially from that championed by the writers of half a generation ago. This management of the patient is nothing else but a continuous piloting between the devil and the deep sea. On the one side the greater intensity degree of the sugar output, on the other side the re-appearance or augmentation of the ketonosis.

However, I am certain that a mild ketonuria is much less of a danger signal than we are wont to believe. Many of my patients exhibited this phenomenon for months and months, without noticeable injury to their general well-being. If I have to choose between the production or the continuation of a mild ketonuria and the suppression of a high-degree glycosuria, I never hesitate to counterwork the latter. It is true enough that the inaugural fast will often work wonders, but even in the successful cases this influence will not endure for a protracted period. Other fast-

ing periods should be intercalated; in some cases when called for, in others as a routine practice.

Taking it all in all, the food intake at the end of the third week following the completion of the inaugural fast (when the provisional determination of the carbohydrate tolerance has been accomplished) is a fair example of the subsequent dieting in the severe forms of diabetes.

Subsequent fasting periods

Cases under observation from the beginning of the inaugural fast until the end of the third week of graded undernutrition, having shown no urinary sugar since the second or third day of the fast, need not be submitted to any subsequent fast during this period. At the end of the fourth week, however, when the patient is receiving in the neighborhood of forty calories per day and kilogram of body weight, a fasting period of about thirty-four hours should be interpolated. Weekly thereafter, so long as he receives from thirty-five to forty calories per day and kilogram, the patient should undergo a fast of the same length. The fast should be taken under this condition even in case the urine of the past week should have proved sugar-free.

I generally let the patient start his thirty-four hours' fast in the afternoon of Saturday (half-holiday) and let him continue it until Sunday evening. He omits the midday and evening meals on Saturday and the breakfast and midday meals

on Sunday. At about five or six o'clock on Sunday evening I let the patient have a light meal composed of oysters or broth, two or three eggs, some green salad, spinach and asparagus. On Monday morning, after having had his usual breakfast, he is ready to attend to his business affairs until the following Saturday afternoon, when the same routine is going to be repeated.

In some cases a two-days' fast is necessary to regulate the metabolic unbalance. These two-days' fasts may also be submitted to in periodic intervals, and it may be necessary to intercalate two-days' fasts every week. However, more than three or four times in succession fasts of that duration should not be ordered as the patients will lose weight and strength too rapidly.

Every fast day, of course, lowers the patient's weight. With from thirty to forty calories per day and kilogram of body weight the diabetic individual may just maintain his own. The intercalated thirty-four hours fasting will invariably reduce his weight for not less than one to one and a half kilograms (two to three pounds). As a matter of fact a loss of from three to four kilograms (six and one-half to almost nine pounds) is not rare. Patients holding their own will make up this loss almost entirely before the next weekly fast. However, there must be a limit in the decline in body weight, and hence also in the number of fast days.

Some recent writers have made a virtue of

necessity, maintaining that the loss of weight is a boon for the diabetic patient. This, as has already been shown, is true to a certain extent so far as the mild cases of diabetes, especially those occurring in association with obesity, are concerned. Severe cases of diabetes, on the other hand, should undergo fasting and undernutrition out of dire necessity only. They must live, for the time being, in conformity with the restricted intake, but they should lose as little as possible in body weight. In severe diabetes, decline of body weight goes in nearly every instance hand in hand with decline in body strength and systemic resistance.

It will be found that in the course of time complete fasting periods cannot be intercalated any longer in any of the cases. They will debilitate the patient more than they will detoxicate him, and they cannot be continued sufficiently long so that in advanced instances the urinary sugar can be any longer materially reduced. As every therapeutic measure so fasting in diabetes has its natural limits.

However, while fasting as such may no longer be applicable (and for obvious reasons this will occur sooner in private than in institutional practice), undernutrition may be resorted to in those stages of severe diabetes in which fasting is no longer of demonstrable value. Here the food intake of the second or the first week after the inaugural fast may be of service.

Water during undernutrition

Besides the liquids pointed out in the various dietaries the patient during the period of undernutrition may take as much water as is consistent with a good gastric digestion and general comfort. As soon as the sugar output has materially diminished, it will be found that the patient has little inclination to drink more than small amounts of water. When this is the case the patient must be made to drink water, that is to say he should imbibe not less than one liter during the twenty-four hours. Aerated waters are apt to cause gastro-intestinal and even vesical distress for which reasons they should not be permitted in the general run of the cases.

When the patient is in a state of undernutrition he should take but small quantities of water at any one time. Each drink should not exceed one hundred cubic centimeters (three ounces). Before the night's rest especially, the water should be taken sparingly. Chopped ice is a very good form in which to carry water into the system.

Evacuation of the bowels

The movements of the bowels during the periods of undernutrition are nearly always sluggish. Assistance has to be rendered by some artificial means. An enema is often all that is needed; one of soap-suds and oil is to be preferred. It must be administered daily, as a rule.

Epsom salts may render good service in instances where kidney disease can positively be excluded. Its dose must presumably be increased every few days. It should be administered daily.

In the long run phenolphthalein or aloin in combination with some carminative should be prescribed. These preparations are best given at bedtime.

Castor oil in sufficiently large doses should be ordered to be taken once or twice each week if the intestinal evacuations are not as copious as they ought to be after the exhibition of aforementioned expedients.

Rest during undernutrition

It is imperative that the diabetic patient in the state of undernutrition has a good deal of rest. Rest in bed is, of course, the best. Some diabetics need considerably more rest than others. However, twelve hours in bed during the twenty-four hours should be the minimum. Many diabetics get along with very little sleep; they hate the bed. Sedatives and hypnotics should not be administered to such patients, but they must learn to rest their bodies and brains without sleeping. Mild exercise like walking is permissible if the patient's strength allows it.

IV

FAILURES

Thus far detailed descriptions were given of the technic of the fast in severe forms of diabetes, of the breaking of the fast, and of the subsequent graded system of undernutrition. Occasionally only allusion has been made that the sailing is not always an easy one, and that there are cases unsuccessfully treated by this combination method.

It has already been stated that the overwhelming majority of diabetics need not submit to a dietetic juggling that has neither any *raison d'être* in the ordinary diabetic economy, nor will or can or is even expected to do more good than is accomplished by the approved methods of diabetes therapy. The advanced and severer forms of diabetes alone—forming about twenty per cent. of all the cases of the affection—are those that should be submitted to the prolonged fasting-undernutrition method. Of these considerably more than half are markedly benefited by this plan of treatment, about one-fifth of the patients are not influenced by it one way or the other, and the general condition (and sometimes also the glycosuria and ketonuria) becomes unquestionably aggravated in about five per cent. of the instances.

Peremptory failures

For the present it may suffice to know that many of the ultimate successful cases may prove failures for a time on account of a wrong technic or for some other reason. However, there are always some patients—a comparatively small number, it is true—who are and remain peremptory failures.

Now, what is a peremptory failure? Among the peremptory failures I count those cases in which an existing glycosuria on prolonged fasting does not decrease or disappear altogether, and in which the ketonuria does not diminish in the ratio in which body weight and vigor decline. Cases in which the glycosuria disappears while the ketonuria persists I do not classify as peremptory failures, as the majority of the diabetic cases with ketonosis treated by prolonged fasting still show evidence of acetonuria. (As a matter of fact, however, the ketone content is, as a rule, more or less diminished after a fast of from four to six days.) Moreover, there are cases which are free from ketonuria on a certain day of the fast and which again exhibit acetone during the following twenty-four hours. And again, there are cases in which a prolonged fast has caused the disappearance of ketonuria, while in the same patient, a fast of even greater length a month later failed to accomplish this feat.

It stands to reason that cases can only be counted among the peremptory failures after

various attempts at protracted therapeutic fasting have been made. The cases do not bear the imprint of peremptory failures upon their foreheads. If they would, some useless treatment, not to say suffering, could be avoided. A number of writers following the lead of v. Noorden assert that in the really severe forms of diabetes the loss of weight sustained on a fast day is rapidly made up or considerable increase even is obtained, on the addition of one hundred grams carbohydrate to the food. Every clinician with some experience in the treatment of diabetes knows that this is by no means the case in the general run of the severe cases, that it is nearly always an impossible task to make the diabetic affected with a grave type of the disease regain what he has lost through restricted diet in general and fasting in particular; and that the clinician can consider himself rather lucky if he is able to avert any further decline of his patient's physical condition.

Cases proving peremptory failures not only belong to this category on account of the unimproved urinary condition, but also—and this is the crux of the entire question—on account of the aggravation of the peculiar physical circumstances of the patient. While autotoxic phenomena play a certain rôle in advanced or severe forms of diabetes, they, by no means, dominate the entire clinical picture. The peculiar muscular weakness of the diabetic, the grave lassi-

tude culminating in listlessness, the entire want of emotion and the absence of "the will to live" are symptoms of undernutrition and not of auto-intoxication. In grave diabetes danger from starvation is just as much a possibility as is danger from autointoxication, and, under existing circumstances, one cannot readily differentiate between the beginning of the one and the termination of the other.

Cases of this sort should, of course, not be subjected to prolonged fasting or undernutrition. The occasional omission of a few meals in order to at least reduce the degree of the glycosuria may be tried. The reduction of the starch intake to a minimum must never be carried out for longer than a day or a day and a half. In some of my cases I have succeeded tolerably well for a time by withdrawing the more substantial starch carriers every third day, that is by placing the patients on a diet consisting of yolks, bacon, whiskey, spinach and lettuce, and keeping them in bed during this time. In a very few cases I could even intercalate one of these restricted diet days every other day. In other instances, however, it is not permissible to intercalate one of these restricted days oftener than every fifth, sixth or seventh day without doing some real harm to the patient. At any rate, many of these cases which are out-and-out failures at fasting and continued starvation may get along in comparative comfort if less treatment is applied to

the urinary features and more attention given to the physical individuality and idiosyncrasies of the patient.

Rule-of-the-thumb measures should certainly not hold sway in the treatment of the severe forms of diabetes. They should be tried, to be sure, but must not be adhered to if they have proved useless or harmful in the particular instances.

Apparent failures

Apparent failures are not real failures. Many of the finally successful cases are failures in one way or the other at the first attempts of the fasting-undernutrition treatment. Blame for this must mainly be given to two causes, viz.: *first*, the lax manner in which the fasting-purging-rest program is prescribed and performed, and, *second*, the too firm adherence to the graded dietaries during the period of undernutrition.

Ad 1.—In order to adjudge the severe cases of diabetes properly and to treat the suitable instances of the malady successfully, it is absolutely necessary that the attending physician delineate minutely every phase and every step of the fasting period before the treatment is instituted. He must have full confidence in the competence and integrity of the nurse in charge of the case during the inaugural fast. The program must be strictly adhered to (if necessary ten days and longer), so long as the patient does not alarmingly fail in body weight, resistance and

strength. If this be the case, fasting should be discontinued immediately. Another attempt at it may be made two or three weeks later. If the patient exhibits then the same alarming symptoms, fasting must again be stopped. Some months later another trial may be given. If again unsuccessful the case belongs to the peremptory failures and should not be treated by this method.

Ad 2.—The graded plan of undernutrition as outlined in the foregoing, is of course, only a tentative one. It is designed for the average case after the fasting period. It cannot in the nature of things be a close fit in every case. It is just here where individualization must set in. For one case the first week of the period of undernutrition is too strenuous, for another the graded dietaries follow each other too quickly, for a third the period of undernutrition is too extended, and for a fourth and a fifth this or that is not especially suitable.

The various untoward phenomena have to be met as they arise. If in a case the daily intake of food appears to be too small to keep body and mind together—a not very rare occurrence in the first week after the fast—a somewhat greater amount may be given. In many cases the skipping of three or four days of the scheduled dietary of the first week makes no difference in the urinary condition and is of distinct advantage to the general well-being of the patient. In other

words, the first week of undernutrition may often be contracted to three or four days. However, if sugar reappears, a fast day should at once replace the third, fourth, etc., day, as the case may be, after which one has to start again with the dietary of the first day following the inaugural fast.

In case the diet of the second week of undernutrition should be productive of urinary sugar, the food intake of the previous week should be ordered and the patient kept on it until the urine has again been sugar-free for three or four days. If this cannot be accomplished in this manner a few fast days should be intercalated, after which the diet of the first week is again ordered. However, instead of giving this diet for one week only it may be necessary to continue it for two weeks or longer. During the time this low diet is taken the patient must rest in bed for from fourteen to eighteen hours every day.

In case a glycosuria should supervene in the third week after the fasting period, a fast day should at once be intercalated and the patient be maintained on the diet of the second week of undernutrition. If this should not render the urine sugar-free, another fast day should be intercalated and the diet of the first week of undernutrition given thereafter. Following this the food of the second week of undernutrition is given for two weeks or longer. When the urine is then free from sugar, the diet of the third

week of undernutrition may be permitted. In short, whenever sugar reappears in the urine, the patient may be rendered sugar-free by fasting and kept sugar-free by the prolonged pursuing of the diet of the week of undernutrition preceding the week during which the sugar had again been excreted.

None of the apparent failures are real failures. In nearly every pertaining case it is only a question of time that the desired object will be attained. True, in some of these cases the carbohydrate tolerance will always remain extremely low. In such cases fats, proteins and vegetables with low carbohydrate content—the diet of the second week of undernutrition with additional fats and proteins—must be given for long-continued periods.

V

PHYSICO-THERAPEUTIC MEASURES IN THE MANAGEMENT OF SEVERE FORMS OF DIABETES

With Especial Reference to the Fasting and Undernutrition Periods

Some of the physical methods of treatment are of essential value in instances of grave or advanced diabetes. This is especially the case during the periods of fasting and undernutrition.

Rest

Attention has repeatedly been drawn in previous paragraphs to the fact that the diabetic patient undergoing fasting or undernutrition needs rest and much of it at that. Rest, in fact, is the *sine qua non* in the management of the severe forms of diabetes. It is the most important of all physico-therapeutic measures during the fasting and undernutrition periods. Appertaining details will be found in Chapters II and III of this little book.

The exact amount of rest needed in the individual case must be left to the common sense of the attendant.

External temperature

A high surrounding temperature is conducive to the physical comfort and the decline of the

glycosuria in severe forms of diabetes. Furthermore, fasting and undernutrition are easier executed in a warm than in a cold surrounding atmosphere. One is less hungry in a warm than in a cold climate.

The conclusions are that the fasting-undernutrition plan of treatment promises considerable success if undertaken during the warm seasons of the year, that in winter time the room temperature should be kept at summer heat, and that the bed temperature should be at or near 100 deg. F. for as long a time as the patient can tolerate it.

To obtain the necessary bed-warmth, the hot-water bag or the electric heating pad must occasionally be resorted to. Superheated air or the so-called leukodescent lamp, applied for the usual brief periods, cannot replace the continuously warm surrounding atmosphere, and have no special value as far as the treatment of the severe types of diabetes is concerned. Profuse perspiration, as induced by the sweat or electric bath is, of course, out of the question, as it virtually counterworks the physiological objects which are attained by a moderate surrounding temperature.

During the cold season patients with severe diabetes should wear flannels, at least when out of the house. They may be allowed to be in a very mild perspiration for one or two hours after the midday repast. In winter they should live in well-ventilated rooms heated to from 75 to 85 deg. F. While in bed, these patients should be

well-covered with blankets, and the temperature of the room be lowered to about 65 deg. F.

The utilization of an increased surrounding temperature in the fasting-undernutrition system of diabetes treatment is based upon the following facts and observations:

In a paper read at the Kongress für innere Medizin, in April, 1905, Lüthje brought out the interesting fact that in the grave diabetes of pan-creatomized dogs the surrounding temperature had a decided influence upon the intensity of the glycosuria. This was found to be markedly depressed when the external temperature was high (30 deg. C.=86 deg. F.). He ascribes the influence of the surrounding temperature upon the fluctuation of the sugar excretion to regulatory processes in connection with the body heat, because the experiment animal, exposed to the cold, in the endeavor to maintain its momentary most important function, the retention of its body heat, suddenly excretes large amounts of sugar. The same author also tried to employ high external temperatures for the treatment of human diabetes. He had four diabetics under his observation for from one to one and a half months. Of these, two cases were severe, one mild, and the last one moderately severe. In temperatures varying between 15 deg. and 30 deg. C. (43 to 86 deg. F.) he also noted fluctuation in the sugar excretion, but not as marked as in the dogs. Under the influence of a high external tempera-

ture tolerance (for what is not stated), body-weight and general vigor rapidly increased.

In the discussion of this paper Klemperer pointed out that the experiments of Luthje tended to clear up some hitherto unexplainable facts of clinical experience. It was known to him for a long time, he said, that a cure at Carlsbad in winter did not agree as well with diabetics as one in summer, and that the beneficial influence of bed-warmth and alcohol upon grave cases of diabetes could now also be accounted for. In the same discussion Kühn mentioned the case of a patient living in Java whom he found affected with a severe type of the disease, and which he had already unfavorably prognosticated years ago. In spite of the ingestion of large amounts of carbohydrates (rice, etc.), the patient was still alive, and Kühn concludes that the patient's prolonged life may be attributable to the relatively high temperature of his abode.

Busquet studied the influence of the external temperature on the intensity of the glycosuria in three cases of diabetes. His tests demonstrated conclusively that the glycosuria constantly decreased when the temperature surrounding the patients was warm, while it increased when it was cold. He never placed his diabetics in temperatures above 20 deg. C. (68 deg. F.), but his clinical findings tallied with those of Luthje in every respect.

At a discussion at the meeting of the British

Medical Association in 1908, on diabetes in the tropics, attention was drawn to the frequent occurrence of glycosuria and diabetes in the natives of India, and to the fact that acute cases are rather infrequently met with in the tropics, where the diabetic is not emaciated and his appetite less abnormal. Sir Havelock Charles made the interesting statement that the progress of the diabetic process in the East Indian is often slower than in the European, and that the disease may last twenty-five years.

The foregoing are practically all the references in regard to the influence of the external temperature on the degree of the glycosuria and the course of diabetes which I could gather from a brief search of the literature. I have known from personal experience for a number of years that diabetes occurring in certain districts of Central and South America, irrespective of a diet very abundant in carbohydrates, often runs a protracted and very mild course. This is also the case to some extent with the diabetic affection in certain localities of our own South. A patient from Mississippi, since dead from old age (he died in the North), assured me that he has had diabetes for over thirty years. From a number of diabetics, who have repeatedly come from the South to consult me, I know that they lost weight and excreted large amounts of sugar when they were in New York for a few weeks, but that their body weight increased and the glycosuria de-

creased soon after they had left the flesh pots of the North for the hot corn and sweet potatoes of their Southern homes. Furthermore, it is a well-known fact that rest in bed is of beneficial influence in many instances of grave diabetes; the good effect thus obtained is, however, universally and solely credited to the rest and not to the bed-warmth.

Without intending to enter into statistical details as regards my own observations concerning the question of surrounding temperature and the course of glycosuria, I wish to offer my conclusion on this occasion: (1) The external temperature exerts a distinct influence upon the intensity of the diabetic glycosuria, inasmuch as this is depressed by warmth and raised by cold. (2) A temperature of from 80 to 90 deg. F. of the surrounding *dry* air may be employed therapeutically to reduce the degree of a diabetic glycosuria. (3) A surrounding temperature of from 80 to 90 deg. F. may *per se* reduce the sugar output by from one-fourth to one-third. (4) A warm external temperature does not materially influence the ketonuria concomitant with grave cases of diabetes. (5) A warm external temperature has no noticeable influence on mild cases of diabetes, but exerts a salutary effect on moderately severe and severe instances of the malady. (6) The salutary effects of a warm surrounding temperature on the diabetic organism, besides the reduction of the glycosuria, consist of the production

of an increased tolerance for carbohydrates and proteids; the increase of body-weight, resistance, and vigor; the lessening of gastro-intestinal disorders, and the subduing of neuritic and angio-sclerotic pain.

Massage

A mild massage of the limbs—five minutes for each limb—may be daily practised in almost every diabetic undergoing the fasting-undernutrition treatment. In many cases the procedure may be repeated on the same day.

In cases after the fasting period in which there occurs much flatulence or in which there is a persistent non-inflammatory gastro-intestinal disturbance such as a paretic condition of the intestine, visceral ptosis (especially gastropptosis or coloptosis), congestive states, etc., general abdominal massage for from fifteen to twenty minutes every day is usually of marked benefit.

While massage is but an accessory measure in the treatment of the severer types of diabetes, it should be employed whenever the patient's condition permits.

Among the physiological effects of massage the mechanical and thermal results occupy first place. The mechanical effects supervene rapidly. Cellular activity becomes increased and there ensues an acceleration in the movement of blood and lymph vessel contents. Massage causes an increase of the temperature of the manipulated

limb. The temperature of the whole body may also be elevated which is especially the case after abdominal massage. Increased body heat causes diminution of the glycosuria intensity in nearly every instance. This is also noticeable in many febrile states.

Walking

As soon as the patient receives food yielding about thirty calories per day and kilogram of body weight, provided there be no impediment of the lower extremities, he should be instructed to take short walks on level ground. The length of the walk and its rapidity of necessity depends upon the patient's strength and endurance. He should, however, not exceed one-eighth of a mile at the first attempt, but he may slowly increase his walk to half a mile. As soon as the patient ingests a somewhat larger amount of food so that about thirty-five calories per day and kilogram body weight be furnished, he may take longer walks, that is up to about one mile. It will be found that most diabetics not obtaining a full caloric supply are able to walk from one to two miles every day without any apparent effort. The daily walks may be taken in "broken doses."

It should be remembered that in ordinary walking the work performed by the muscles of the leg is much less than it appears to be, since the swaying motion of the leg, like the swinging of a pendulum, is a passive operation to the greater part.

VI

ILLUSTRATIVE CASES

In the following lines some instances illustrative of the fasting-undernutrition plan of treatment and its results are given. In these case-histories only the data pertaining to the fasting-undernutrition period which are of the most general clinical interest are related.

CASE I.—March 20, 1913. Woman, forty-one years old; widow; has had two children, both dead.

Diabetes had supervened about two years ago; had then weighed 189 pounds. Present weight 145 pounds. Complains of sleeplessness; headaches; extreme weakness and pains in thighs.

The physical examination showed very flabby muscles; rather small liver; blood pressure, systolic, 105 mm. Hg. The urine showed the following features:

Twenty-four hours' amount 2200 c.c.; specific gravity 1030; *glucose* 12 *per cent.*; acetone medium amount; diacetic acid present; ammonia increased; albumin absent; no evidence of renal disease.

The patient was immediately ordered to go to bed and to fast for four days. During this time nothing but weak tea (without milk or sugar) and

water were allowed. The patient was instructed to take a tablespoonful to a tablespoonful and a half of Epsom salts in the evening.

March 24. Patient feels comfortable. *Urinary features*: twenty-four hours' amount 1350 c.c.; specific gravity 1019; *glucose absent, acetone increased*; ammonia stationary.

March 25. Still fasting. Feels well. *Urinary features*: twenty-four hours' amount 1400 c.c.; specific gravity 1019.5; *glucose absent; acetone much increased*; ammonia increased.

March 31. At my office. Feels stronger; slept very well during past week; has only occasional headaches; pains in thighs much improved.

Has been on yolk-protein-green vegetable diet since breaking the fast. Average daily intake twenty calories for each kilogram body weight. Present weight 139 pounds. *Urinary features*: Twenty-four hours' amount 1320 c.c.; specific gravity 1016; *glucose 1 per cent.; acetone decreased*.

April 7. At my office. Feels decidedly better; has uninterrupted night rest; no more headaches; no longer pains in thighs.

Has been on about the same diet, but somewhat enlarged quantities. Average daily intake twenty-five calories for each kilogram body weight. Present weight 143½ pounds. *Urinary features*: Twenty-four hours' amount 1560 c.c.; specific gravity 1020; *glucose 1 per cent.; acetone medium amount*; ammonia decreased.

April 14. At my office. Feels strong and well; slight pain in thighs. The same diet as last week, but had in addition half slice of toast in the morning, half a slice in the evening. Present weight 143½ pounds. *Urinary features:* Twenty-four hours' amount 2800 c.c.; specific gravity 1009; *glucose absent; acetone absent; diacetic acid absent; ammonia normal amount.*

April 21. At my office. States that she "feels grand"; no pain in thighs; uninterrupted night rest; looks very well.

Has been on the same diet, but instead of half a slice had whole slice of toast in the morning and evening. Present weight 143½ pounds. *Urinary features:* Twenty-four hours' amount 2000 c.c.; specific gravity 1017; *glucose absent; acetone trace; no diacetic acid; ammonia normal and stationary.*

April 28. At my office. Looks and feels well; entirely free from pain; sleeps through entire night.

Has been on the same yolk-protein-green vegetable regimen plus three slices of toast per day. Present weight 143½ pounds. *Urinary features:* Twenty-four hours' amount 1800 c.c.; specific gravity 1014; *glucose absent; acetone absent; diacetic acid absent; albumin absent; ammonia decreased.*

May 5. At my office. "Feels fine." Free from pain. Uninterrupted sleep at night.

The same diet as last week. Present weight

145½ pounds. *Urinary features:* Twenty-four hours' amount 1600 c.c.; specific gravity 1010; *glucose 0.1 per cent.; acetone absent; diacetic acid absent.*

May 12. At my office. "Feels as well as ever."

Has had the same diet, but only two slices of toast per day. Present weight 144½ pounds. *Urinary features:* Twenty-four hours' amount 1750 c.c.; specific gravity 1016; *glucose absent; acetone absent; diacetic acid absent; no ammonia increase.*

May 26. At my office. Feels and looks well. Patient thinks she is cured.

The same diet; only two slices of toast daily. Present weight 147½ pounds. *Urinary features:* Twenty-four hours' amount 1750 c.c.; specific gravity 1015; *glucose absent; acetone absent; diacetic acid absent; ammonia in normal amounts.*

June 9. At my office. Feels and looks well.

The same diet; three slices of toast daily. Present weight 146¾ pounds. *Urinary features:* Twenty-four hours' amount 1750 c.c.; specific gravity 1002; *glucose absent; acetone and diacetic acid absent; albumin absent; no microscopic evidence of any renal lesion.*

June 23. At my office. Looks and feels well.

The same diet; three slices of toast daily. Present weight 151½ pounds. *Urinary features:* Twenty-four hours' amount 1750 c.c.; specific gravity 1011; *glucose absent; acetone and diacetic*

acid absent; no albumin; no microscopic evidence of any renal lesion.

Remarks: This case is remarkable inasmuch as there ensued a continuous increase in weight despite a diet which was not only restricted, but the fuel value of which never exceeded thirty calories per day and kilogram of body weight for any length of time.

CASE II.—December 28, 1912. Man, fifty-one years old, single. Theatrical manager. Principal complaint: Weakness.

Diabetes was recognized about eighteen months ago. Was since under treatment; also was in two sanatoria. During this period has had as much as seven per cent. glucose and much acetone. At times the urine was sugar and ketone free. Has lost about twenty-five pounds in weight. The physical examination revealed besides a much emaciated body and flabby muscles nothing that was truly abnormal. The heart was not diseased; systolic blood pressure 140 mm. Hg.; liver not enlarged; no abdominal pressure sensitiveness. Hemoglobin 64 per cent. *Urinary features:* Twenty-four hours' amount 1860 c.c.; specific gravity 1032; *glucose 8.6 per cent.; acetone very large amounts; diacetic acid very large amounts; ammonia excessive amounts; indican absent; no microscopic evidence of renal disease.*

Diet: The patient was ordered to take a prolonged fast under the supervision of an experienced nurse. Water, tea and Hunyadi water were

to be taken during the fast. After the third fast day 40 c.c. alcohol (80 c.c. whiskey) was permitted during the twenty-four hours.

Dec. 29. Feels well. Not very hungry. *Urinary features*: Twenty-four hours' amount 1580 c.c.; specific gravity 1020; *glucose absent*; acetone and diacetic acid very large amounts.

Dec. 30. Does not feel hungry. *Urinary features*: Twenty-four hours' amount 1300 c.c.; specific gravity 1017; *glucose absent*; acetone and diacetic acid very large amounts. The purgative upset the patient considerably.

Dec. 31. Feels more comfortable. *Urinary features*: Twenty-four hours' amount 1220 c.c.; specific gravity 1014; *glucose absent*; acetone and diacetic acid large amounts.

Jan. 1, 1913. Feels comfortable. *Urinary features*: Twenty-four hours' amount 900 c.c.; specific gravity 1008; *glucose absent*; acetone and diacetic acid large amounts. Patient was permitted to take whiskey and in addition 60 grams green vegetables with low carbohydrate content.

Jan. 2. Feels comfortable. *Urinary features*: Twenty-four hours' amount 980 c.c.; specific gravity 1018; *glucose 0.83 per cent.*; acetone and diacetic acid large amounts.

Jan. 3. Fasting.

Jan. 4. Fasting.

Jan. 5. Fasting. Feels comfortable, but hungry. *Urinary features*: Twenty-four hours' amount 1200 c.c.; specific gravity 1013; *glucose*

absent; acetone and diacetic acid large amounts.

The patient was advised to remain in bed most of the time. Diet: Exclusion of all starches; limited quantity of proteins.

Jan. 7. Feels hungry. *Urinary features*: Twenty-four hours amount 2000 c.c.; specific gravity 1017; *glucose absent*; acetone and diacetic acid large amounts.

Apenta water in large doses was ordered to be taken.

Diet: Green vegetables; fifteen yolks; albumin of seven eggs.

Jan. 8. Hunger is appeased. *Urinary features*: Twenty-four hours' amount 1650 c.c.; specific gravity 1022; *glucose absent*; acetone and diacetic acid unchanged in amount.

Jan. 9. Feels well. Same diet. Present weight 117½ pounds. *Urinary features*: Twenty-four hours' amount 2350 c.c.; specific gravity 1018; *glucose absent*; acetone large amounts.

Jan. 10. Patient was out of the house all day yesterday. *Urinary features*: Twenty-four hours' amount 2300 c.c.; specific gravity 1014; *glucose absent*; acetone very small amount; diacetic acid *absent*.

Jan. 11. Feels well. *Urinary features*: Twenty-four hours' amount 2000 c.c.; specific gravity 1015; *glucose absent*; acetone small amount; diacetic acid *absent*.

Diet: Eighteen yolks and eight egg whites daily in addition to green vegetables.

Jan. 12. Feels well. *Urinary features*: Twenty-four hours' amount 2200 c.c.; specific gravity 1016; *glucose absent; acetone a trace.*

Jan. 13. Feels well. Present weight 118 pounds. *Urinary features*. Twenty-four hours' amount 2130 c.c.; specific gravity 1020; *glucose absent; acetone and diacetic acid traces.*

Diet: The same; in addition 180 c.c. whiskey.

Jan. 14. Feels somewhat depressed; pronounced phosphaturia. *Urinary features*: Twenty-four hours' amount 2100 c.c.; specific gravity 1024; very turbid; large amounts of earthy phosphates; reaction alkaline; *glucose absent; acetone mere trace.*

Jan. 15. Depressed. Mouth temperature 98 deg. F. Present weight 119½ pounds. *Urinary features*: Twenty-four hours' amount 2100 c.c.; specific gravity 1020; *glucose absent; acetone trace.*

Patient was ordered to discontinue Apenta water for two days.

Diet: Green vegetables; twenty-one yolks; ten egg whites.

Jan. 16. Feels better. Mouth temperature 98.2 deg. F.; pulse 76. *Urinary features*: Twenty-four hours' amount 1830 c.c.; specific gravity 1020; *glucose absent; acetone and diacetic acid absent.*

Since this time the patient has not only held his own, but has gained about three pounds. His urine is sugar-free most of the time; in case he

excretes sugar this does not exceed twenty or thirty grams in the twenty-four hours. Ketones appear but rarely in his urine. In other respects the patient has his ups and downs, but fasting at proper intervals and long-continued periods of undernutrition have enabled him to devote all of his time to business which is a very extensive and exacting one. On Sundays he remains in bed and fasts or has but one meal during the twenty-four hours.

CASE III.—August 31, 1910. Man, forty-nine years old. Has had diabetes for about one year. Complaint: Shortness of breath, especially upon walking. No nocturnal urination. No particular weakness. Decline of sexual desire. Little loss of body weight. *Urinary features:* Twenty-four hours' amount 1800 c.c.; specific gravity 1022.5; glucose 2.5 per cent.; acetone and diacetic acid absent; albumin absent; indican absent; no microscopic evidence of renal disease. The case was considered to be of a mild type and was successfully treated by the usual dietary means.

August 7, 1912. Patient feels and looks well, but the diabetic condition has become aggravated to some degree. He has lost about ten pounds in body weight during the last four months, urinates once or twice during the night, and the twenty-four hours' urine has increased to about 2500 c.c. Patient is sexually entirely impotent. He is living in the South and states that he had a good deal of business worry during the last few

months. The urine, voided at my office, showed a specific gravity of 1024; glucose 8 per cent.; medium amounts of acetone and diacetic acid and increased ammonia output. The urine contained no other abnormal substances or normal substances in abnormal quantity, and there was no evidence of any kidney lesion.

The patient was advised to undergo a therapeutic fast of three or four days' duration. During this period water and tea were allowed. Castor oil was to be taken before starting the fast, and Epsom salt, in purging doses, every day thereafter during the fasting period. Rest in bed was made compulsory.

August 8. Feels well. *Urinary features:* Twenty-four hours' amount 1800 c.c.; specific gravity 1020; glucose 2.5 per cent.; acetone and diacetic acid medium amounts.

August 9. Feels well. *Urinary features:* Twenty-four hours' amount 1680 c.c.; specific gravity 1015; *glucose 0.2 per cent.; acetone trace; diacetic acid absent.*

August 10. Feels well; is not particularly hungry. *Urinary features:* Twenty-four hours' amount 1400 c.c.; specific gravity 1013; *glucose absent; acetone trace; diacetic acid absent.*

August 11. Feels comfortable. *Urinary features:* Twenty-four hours' amount 1460 c.c.; specific gravity 1012.5; *glucose absent; acetone and diacetic acid absent.*

The patient was kept on the graded plan of

undernutrition for three weeks. Starch in the form of toast was permitted thereafter, at first tentatively, *i.e.*, half a slice twice a day. Later on two and three slices a day were tolerated. He can take now four or five slices of bread a day. For one week each month he pursues a fat-protein-vegetable diet (excluding cream, butter, cheese and vegetables containing more than five per cent. available starch).

CASE IV. October 16, 1913. Man, 52 years old; Member of Congress. Has diabetes for about ten years. Complains of extreme weakness.

A physical examination shows a very dry skin; fat heart; enlarged liver and stomach. The latter is also ptotic. There is no pressure point sensitiveness. The urine exhibits a specific gravity of 1030; glucose 12.5 per cent.; some acetone and diacetic acid; no albumin; no indican. It shows no microscopic evidence of renal disease.

As the patient had much declined in body weight and vigor since he was last seen by me, a three or four days' fast was proposed. The patient readily consented.

October 21. First fast day. Feels very well. *Urinary features:* Twenty-four hours' amount 2150 c.c.; specific gravity 1027; *glucose* 2.5 per cent.; acetone trace.

October 22. Feels comfortable. Not hungry. *Urinary features:* Twenty-four hours' amount 1460 c.c.; specific gravity 1020; *glucose* 0.25 per cent.; acetone somewhat increased.

October 23. Feels well. Not particularly hungry. *Urinary features:* Twenty-four hours' amount 1320 c.c.; specific gravity 1014; *glucose absent; acetone considerably increased; diacetic acid present; albumin trace.*

October 24. Feels well, but rather weak. *Urinary features:* Twenty-four hours' amount 1550 c.c.; specific gravity 1014; *glucose absent; acetone stationary in amount.*

October 25. Has lost over nine pounds during the fast. Feels a little weak, but very well otherwise. Institution of undernutrition.

October 27. Feels stronger. *Urinary features:* *Glucose absent; acetone very small amount.*

October 31. Feels very well. *Urinary features:* *Glucose absent; acetone trace.*

November 5. Feels well. *Urinary features:* Twenty-four hours' amount 1700 c.c.; specific gravity 1015; *glucose absent; acetone small amount.*

Diet: Undernutrition regimen of end of second week; in addition one slice of toast in the morning and evening.

November 7. *Urinary features:* Twenty-four hours' amount 1750 c.c.; specific gravity 1020; *glucose absent; acetone trace.*

Diet: The same, excepting two slices of toast in the morning and one slice in the evening.

November 10. Feels very well. Physical examination reveals nothing abnormal of import. *Urinary features:* Twenty-four hours' amount

1900 c.c.; specific gravity 1018; *glucose absent*; *acetone absent*.

Diet: Undernutrition, end of second week; in addition two slices of toast in the morning and evening.

November 12. *Urinary features*: Specific gravity 1018; *glucose absent*; *acetone absent*.

The patient attends to his arduous duties as a member of Congress and at home. He abstains entirely from food for about thirty-six hours every month. For one week every month, following the fast, he adheres to the diet, prescribed for the last days of the second week of the period of undernutrition. At this time he transacts most of his business while he is resting in bed. Despite the frequent fasting and periods of undernutrition the patient has lost not more than about sixteen pounds in thirty-four months.

CASE V.—October 9, 1913. Woman, fifty-two years old; one child. Has had diabetes for fifteen years. States that she was operated upon seventeen times (fourteen times by one of the most renowned American gynecologists) for genito-urinary diseases. Present weight 161½ pounds.

The physical examination reveals a distinct murmur (presystolic), and a distinct accentuation of the second aortic sound. Abdomen is pendulous; liver somewhat enlarged and ptotic. There is an umbilical hernia. No pressure point sensitiveness. The skin is full of "liver spots"

of rather recent production. The urine is voided in daily amounts of about 2000 c.c.

October 10. *Urinary features:* Twenty-four hours' amount 1900 c.c.; specific gravity 1024; glucose 7.14 per cent.; acetone medium amount; diacetic acid present; very turbid; microscopically there were distinguished epithelia of lower genito-urinary tract and from renal pelvis; pus corpuscles; ammonium urate crystals; colon bacilli.

The case was considered to be one of an advanced, though not of a serious type of diabetes, and a fast of three days' duration was ordered.

October 14. After the fast. Feels well, but is very recalcitrant. Present weight 158 pounds. *Urinary features:* Twenty-four hours' amount 1200; specific gravity 1019; *glucose* 0.416 per cent.; acetone a trace; diacetic acid absent.

Diet: Undernutrition, first, second and third weeks (yolks, proteins, green vegetables).

November 4. Feels very well. Present weight 159 pounds. *Urinary features:* Twenty-four hours' amount not stated; specific gravity 1024; *glucose* absent; acetone a trace.

The treatment was continued by the family physician. Despite a rather large excretion of sugar this case is not of grave character. It is of long duration—fifteen years—at the start of the fasting-undernutrition plan of treatment. The urinary anomalies yielded readily, but the pa-

tient had to be watched very carefully as she had no self-control whatever.

CASE VI.—September 15, 1914. Man, fifty-five years old; attorney-at-law. Diabetes was first recognized in May, 1914.

Has lost about thirty pounds in body weight since the onset of the affection. Present weight 112¾ pounds. Complains of weakness, rhinitis and extreme dryness of the mouth (it could be demonstrated later on that this dryness was part and parcel of a very pronounced ketonosis). The physical examination revealed a dry skin and dry mucous membranes, a slightly dilated heart, but no other cardiac irregularity of any kind. The liver was somewhat enlarged, more so toward the median line. It presented no abnormality in hardness, etc. The stomach was crowded toward the left; the cecum was somewhat inflated. No pressure point sensitiveness anywhere.

Urinary features: Twenty-four hours' amount 2200 c.c.; specific gravity 1026; *glucose* 12.5 per cent.; acetone and diacetic acid very large amounts; albumin medium amount; microscopic evidence of sclerotic kidney.

Treatment: Prolonged fasting; allowed during the fast water, tea and whiskey (60 c.c. per day); castor oil.

September 17. Feels about the same as before starting the fast. *Urinary features:* Twenty-four hours' amount 2000 c.c.; specific gravity 1022;

glucose 8.33 per cent.; acetone and diacetic acid very large amounts; albumin medium amount.

September 18. Feels weak, but comfortable. *Urinary features:* Twenty-four hours' amount 1750 c.c.; specific gravity 1015; glucose 3.57 per cent. (64.68 grams); acetone unchanged in amount; diacetic acid very large quantity.

September 19. General condition unchanged. *Urinary features:* Twenty-four hours' amount 2000 c.c.; specific gravity 1010; glucose 1.25 per cent.; acetone and diacetic acid unchanged in amount.

September 20. Feels comfortable. *Urinary features:* Twenty-four hours' amount 1750 c.c.; specific gravity 1008; glucose 0.9 per cent.; acetone unchanged; diacetic acid large amount.

September 21. Feels stronger. *Urinary features:* Twenty-four hours' amount 1500 c.c.; specific gravity 1007; *glucose absent; acetone decidedly decreased;* diacetic acid traces.

Patient refused to fast longer. Undernutrition (yolks-green vegetables), about 800 calories per day, called forth a glucose output of from 15.65 grams to 85 grams per day during the following week. The ketone excretion was increased, but did not reach then the values which obtained before the fast. Despite the continued excretion of glucose and ketones the patient had reached about 120 pounds in weight on November 4.

Patient could not again be persuaded to submit to another fast of the same duration. He would

execute any other dietary stunt. This, however, proved without avail. Some time ago he wrote me that his urine was free from sugar and acetone. This, I doubt, unless he has submitted to the fasting-undernutrition treatment since I last saw him.

CASE VII.—May 24, 1906. Woman, fifty-three years old; two children.

Diabetes was diagnosed some years ago. Principal complaint when first seen by me was pruritus vulvæ. Former weight 190 pounds; present weight 160½ pounds.

The case was a rather mild one, belonging to the type of diabetes of the obese. For more than eight years the diet of the patient was not rigid, the daily amount of permitted carbohydrate varying between 100 and 150 grams. Slowly the carbohydrate tolerance declined and ketonuria, at first more or less intermittent, ensued. In meantime an ulcer malum on the right foot and some minor gangrenous processes, due no doubt to obliterating, atherosclerotic changes, had formed. For these various reasons the patient was instructed to undergo a fasting-undernutrition treatment in November, 1914.

November 4. (Before fast) *Urinary features*: Twenty-four hours' amount 2200 c.c.; specific gravity 1028; glucose 3.5 per cent. (77 grams); acetone and diacetic acid medium amounts; ammonia much increased.

The patient utilized a servant as a nurse dur-

ing the fast. Tea, water and Epsom salts were taken in the usual amounts. Alcohol was refused by the patient.

November 8. After three days' fast. Patient feels well; is not particularly hungry. *Urinary features:* Twenty-four hours' amount 1200 c.c.; specific gravity 1020; glucose one per cent. (12 grams). Acetone and diacetic acid small amounts.

Slough of gangrenous processes decidedly less (proper local treatment was applied all the time).

November 11. After six and one-half days' fast. Feels very comfortable; not hungry and not "really weak." *Urinary features:* Twenty-four hours' amount 1000 c.c.; specific gravity 1020; *glucose absent; acetone and diacetic acid absent.*

The patient was thereafter kept on the afore-described graded plan of undernutrition. The urine remained sugar and ketone-free for a number of months. Weekly fast days were intercalated for some time. During the summer of 1915 the patient relaxed somewhat in her regime with the result that the pathological urinary features became aggravated. The patient has since been subjected to a series of fasting periods; the outcome proved always beneficial. The various gangrenous processes have entirely healed. (The patient has received for the latter not alone the proper antiseptic applications and surgical treatment, but also footbaths of sodium salicylate, superheated air, etc.) The healing of these gan-

grenous mortifications must be, in part at least, ascribed to the dietary influences.

CASE VIII.—March 24, 1915. Woman, forty years old; two children.

Diabetic symptoms are present for about two years. Former weight 145 pounds; present weight 115 pounds. Is lean, but quite energetic. Attends to her affairs, but is often very tired and exhausted. Has noted decline of sexual appetite. Complains especially of interstitial gingivitis. The physical examination revealed an emaciated body with extremely dry external integument. No cardiac lesion. Blood pressure, systolic, 120 mm. Hg. Liver somewhat enlarged. No pressure point sensitiveness anywhere. Deep reflexes slightly exaggerated. Wassermann negative. The urine showed the following features: Twenty-four hours' amount 2100 c.c.; specific gravity 1034; glucose 20 per cent. (420 grams); acetone and diacetic acid large amounts; ammonia increased; albumin absent; no microscopic evidence of renal disease. Patient is under a very liberal diet which has been prescribed by the family physician. Partakes of plenty of milk, cream, bread and fruit. I ordered a fat-protein-green vegetable diet plus two slices of toast per day.

March 29. Patient feels somewhat stronger. Present weight 109 pounds. *Urinary features:* Twenty-four hours' amount 2000 c.c.; specific gravity 1030; glucose 5 per cent. (100 grams);

acetone much reduced; diacetic acid unchanged; ammonia stationary; no indican; no albumin.

Diet: The same, but three instead of two slices of toast per day.

April 1. Is somewhat stronger. *Urinary features:* Twenty-four hours' amount 1920 c.c.; specific gravity 1040; glucose 10 per cent. (192 grams); acetone increased; diacetic acid unchanged; ammonia slightly increased; albumin and indican absent.

Diet: The same, excepting toast. As only carbohydrate rice, 30 grams, boiled, in morning, 30 grams in evening.

April 8. Feels very weak. *Urinary features:* Twenty-four hours' amount 2000 c.c.; specific gravity 1030; glucose 5 per cent. (100 grams); acetone decreased; diacetic acid absent; ammonia much decreased; albumin and indican absent.

Diet: The same; increased amount of yolks. To add sodium bicarbonate in large doses.

April 14. Looks and feels better. Has passed large amounts of earthy phosphates during last few days. *Urinary features:* Twenty-four hours' amount 2100 c.c.; specific gravity 1030; glucose 5 per cent.; acetone much increased; diacetic acid present; ammonia increased; indican and albumin absent. Yolks and sodium bicarbonate without avail in reducing ketone substances.

Diet: The same, however, instead of rice oatmeal in the same quantity.

April 21. Feels better and much stronger. Present weight 107½ pounds. *Urinary features:* Twenty-four hours' amount 1850 c.c.; specific gravity 1030; glucose 5 per cent. (92.5 grams); acetone large amount; diacetic acid medium amount; ammonia increased; indican and albumin absent.

Diet: The same; 30 grams oatmeal in the morning; 30 grams in the evening.

April 28. Feels better and stronger. Has taken much water and perspired profusely (hot weather); has ingested from seventeen to twenty yolks (six whole eggs) per day. Present weight 110½ pounds. *Urinary features:* Twenty-four hours' amount 3000 c.c.; specific gravity 1034; glucose 7 per cent. (210 grams); acetone large amounts; diacetic acid unchanged; ammonia increased; albumin and indican absent.

Diet: The same, including the large amount of yolks. Under this plan of treatment the patient held her own until the beginning of October, when her weight had declined to 107 pounds. The urine during this period always contained between three and eight per cent. of sugar, and was not ketone-free on a single occasion. After oatmeal as "sole carbohydrate" had been taken for some months it was replaced by potatoes. When potatoes as the sole carbohydrate were ingested (three per day) the glucose output never exceeded 5 per cent., and the twenty-four hours' urine did not exceed 1800 c.c. Hence never more

than 90 grams glucose were excreted on any of the potato days.

In order to render the patient sugar-free and, if possible, also ketone-free, a fasting period under the supervision of an experienced attendant was ordered on October 10. The patient was instructed to go to bed and stay there during the entire period of fasting, to take medium doses of Epsom salts every day, and water or tea when desired. Whiskey not to exceed 60 c.c. per day was allowed to be taken in broken doses. It was advised to take resort to the electric pad in case the patient felt slightly chilled.

October 11. Patient is comfortable and does not complain of hunger. Has slept three hours in the afternoon and nearly through the night. Had two copious movements after taking magnesium sulphate. Rectal temperature at 8 A.M. 99 deg. F.; pulse 90; respiration 20; rectal temperature at 4 P.M. 98.6 deg. F.; pulse 92; respiration 20. Intake of liquids during twenty-four hours 1200 c.c. *Urinary features:* Twenty-four hours' amount 630 c.c.; specific gravity 1018; glucose 1.86 per cent. (11.71 grams); acetone, diacetic acid and ammonia diminished.

October 12. Patient is comfortable and does not complain of hunger. Has slept well. Rectal temperature at 8 A.M. 99.2 deg. F.; pulse 100; respiration 20; rectal temperature at 4 P.M. 98.4 deg. F.; pulse 88; respiration 20. Intake of liquids during last twenty-four hours 900 c.c.;

urine twenty-four hours amount 630 c.c. *Other urinary features:* Specific gravity 1015; *glucose absent; acetone and diacetic acid absent; ammonia diminished.*

The fast was continued for four days longer, during which time the urine remained free from sugar and ketones. The plan of undernutrition treatment was started on October 17. The urine on this date showed neither sugar nor ketones. On October 24, when the urine was still free from sugar and ketones, the second week of undernutrition was started. The food which the patient then obtained yielded from about 1000 to 1100 available calories per day.

October 27. The patient feels well, but has slightly lost in weight. The urine *is free from sugar and ketones.*

November 24. After a two-weeks' stay at Atlantic City, during which time the patient had more or less overstepped the undernutrition boundaries, she feels and looks well. *Urinary features:* Twenty-four hours' amount 1600 c.c.; specific gravity 1029; glucose 2.86 per cent. (45.76 grams); acetone and diacetic acid medium amounts; albumin absent.

Fasting period ordered. Tea and water allowed; alcohol permitted in amounts of from 40 to 60 c.c. (from 80 to 120 c.c. whiskey) per day. Epsom salts. No attending nurse.

November 28. After fasting three and one-half days. Feels well and not hungry. *Urinary*

features: Twenty-four hours' amount 850 c.c.; specific gravity 1015; *glucose absent*; *acetone trace*; *diacetic acid absent*.

December 1. After six days of fasting. Feels well. *Urinary features:* Twenty-four hours' amount 800 c.c.; specific gravity 1015; *glucose absent*; acetone small amount; diacetic acid small amount.

December 3. After eight days of fasting. Patient feels well. *Urinary features:* Twenty-four hours' amount 800 c.c.; specific gravity 1012; *glucose absent*; acetone slightly increased; diacetic acid slightly increased; ammonia increased; albumin and indican absent.

The following *diet* (undernutrition) was ordered: Breakfast—two soft boiled eggs, cup of tea without milk or sugar; 10 A.M.—15 c.c. whiskey; luncheon—two cups of bouillon, soup-plateful of spinach or string beans, green salad; 2 P.M. 15 c.c. whiskey; 4 P.M. cup of tea without milk or sugar; supper—bouillon, one egg, soup-plateful of green vegetables, green salad; 8 P.M. 15 c.c. whiskey.

December 10. Has strictly complied with diet. Feels well. *Urinary features:* Twenty-four hours' amount 1100 c.c.; specific gravity 1025; glucose 1 per cent. (11 grams); acetone and diacetic acid small amount.

Diet: The same; in addition six more yolks to the daily ration. To walk one-half mile daily if weather permits.

December 17. Looks well. States that she feels very hungry. Present weight 102 pounds. *Urinary features*: Twenty-four hours' amount 1000 c.c.; specific gravity 1020; *glucose absent*; *acetone and diacetic acid absent*.

The patient weighs now in the neighborhood of 111 pounds. She feels very well and energetic and attends to most of her household duties. She is sugar-free most of the time. The few times she excreted sugar during the last eight months the daily amount never exceeded five or six grams. On no occasion has the patient excreted acetone or diacetic acid since her improvement last December.

The patient is now very well able to manage her own case. As a rule she fasts one day every week; occasionally she only fasts for one day every second week. She obtains about thirty calories per day and kilogram. The food is almost entirely absorbable. She never ingests less than ten yolks per day. Besides the starches yielded by green vegetables she also obtains about 30 grams of starch a day in the form of toast. A small amount of water or muskmelon was permitted of late.

This case is one of the most interesting ones which I had the good fortune to observe. For this reason I have reported it somewhat in detail. It clearly demonstrates the great value of fasting and undernutrition in suitable cases. This case was, and probably still is, a very grave instance

of diabetic deterioration. The patient was treated on "new-fashioned," liberal lines by her family physician, which aggravated the disease without any doubt. For nearly seven months I tried to render the patient sugar- and ketone-free by the older methods of dieting without any assured success. Within a few weeks from the start of the inaugural fast the entire type of the affection of this patient seemed to have changed.

CASE IX.—July 28, 1915. Man, thirty-eight years old. Had indigestion for years; obtained life insurance policy two and a half years ago. In December, 1914, had colicky pains and was operated upon for appendicitis. Four days after the operation had a "stroke of paralysis," viz.: face was drawn to one side; had aphonia; could not write with right hand; had frontal headache for two weeks. He could speak in whispers after about two weeks and at about the same time there ensued a corresponding improvement in the right arm. Sugar was found in patient's urine shortly after the operation; acetone was also demonstrated on some occasions. Complains of nocturnal urinations and absence of sexual desire. Speaks very deliberately. Intonation is very monotonous.

The examination showed the following: Heart, weak impulse; no valvular or myocardial disease; slight accentuation of pulmonic and aortic second sounds; blood pressure, systolic, 95 mm. Hg. Liver is somewhat enlarged; freely palpable on

deep inspiration; stomach normal in size; pressure point sensitiveness in lower epigastric region; intestines apparently not abnormal; cecum somewhat enlarged. Deep reflexes normal; anesthetic zones over portions of right arm; coordination and accommodation fair; pupillary reflexes somewhat sluggish. Wassermann test negative; cerebrospinal fluid negative. *Urinary features:* Twenty-four hours' amount 2600 c.c.; specific gravity 1034; glucose 16 per cent. (335.6 grams); acetone and diacetic acid large amounts.

Diet: Fat-protein-green vegetables.

July 31. Feels weak and tired out; nocturnal micturition. *Urinary features:* Twenty-four hours' amount 1800 c.c.; specific gravity 1027; glucose 8.66 per cent. (155.85 grams); acetone and diacetic acid large amounts.

Diet: Unchanged.

August 2. Feels very weak. No nocturnal micturition. *Urinary features:* Twenty-four hours' amount 2000 c.c.; specific gravity 1030; glucose 4 per cent. (80 grams); acetone unchanged in amount.

August 4. Feels very weak; has lost seven pounds since dieting. *Urinary features:* Twenty-four hours' amount 1560 c.c.; glucose 0.25 per cent. (3.19 grams); acetone very large amount; diacetic acid ditto.

August 5. Still weak. *Urinary features:* Twenty-four hours' amount 1500 c.c.; specific

gravity 1016; *glucose absent*; acetone and diacetic acid medium amounts.

August 6. Is stronger, but complains of cramps in epigastric region. *Urinary features*: Twenty-four hours' amount 1620 c.c.; specific gravity 1023; *glucose absent*; acetone and diacetic acid medium amounts.

Diet: Unchanged.

August 7. Feels stronger. Complains of watery stools. *Urinary features*: Twenty-four hours' amount 900 c.c.; specific gravity 1030; glucose 0.5 per cent. (4.5 grams); acetone and diacetic acid medium amounts.

August 9. Feels very weak. Bowels were not evacuated for two days. Examination of the epigastrium and neighboring regions shows nothing abnormal. Liver is somewhat enlarged, especially toward left. This may be due to the head of the pancreas. There is no pressure pain in pancreatic region or anywhere else in abdomen. *Urinary features*: Twenty-four hours' amount 1360 c.c.; specific gravity 1029; glucose 1.25 per cent. (17 grams); acetone medium amount; diacetic acid small amount; albumin and indican absent; no microscopic evidence of renal irritation.

Diet: Unchanged.

August 10. Feels better. *Urinary features*: Twenty-four hours' amount not ascertained; specific gravity 1025; glucose absent; acetone medium amount.

In order to detoxicate the patient and to render him, if possible, ketone-free, a fasting period of four days and complete rest in bed were ordered. During the fast Epsom salts in sufficient doses was to be taken every day. Tea and water were allowed as much as desired.

August 14. After a fast of four entire days. Feels very comfortable; is not particularly hungry; has perspired during the entire time. *Urinary features:* Twenty-four hours' amount 900 c.c.; specific gravity 1020; *glucose absent; acetone and diacetic acid absent;* ammonia normal amount.

Diet: The same as prior to the fast (undernutrition).

August 16. Does not feel very strong. *Urinary features:* Twenty-four hours' amount 1350 and *diacetic acid absent.*

Inasmuch as the patient is compelled to return to his home in Utah within the next forty-eight hours, some toast (half a slice at noon and half a slice in the evening) is added to his diet.

August 17. Feels better. *Urinary features:* Twenty-four hours' amount 1300 c.c.; specific gravity 1025; *glucose absent; acetone and diacetic acid absent.*

Diet: The same, but instead of half a slice one whole slice of toast to be added to the midday and evening meals.

August 18. Feels better. Has gained nearly two pounds in body weight. *Urinary features:*

Twenty-four hours' amount 1320 c.c.; specific gravity 1023; *glucose absent; acetone and diacetic acid absent.*

This case is interesting for the fact that undernutrition could not suppress ketonosis while fasting for a few days freed the system from acetone and its mother substance. The influence of undernutrition was not sufficiently strong and far-reaching. In other instances fasting is apt to aggravate an existing ketonuria.

PART II

SUPPLEMENTARY NOTES ON THE TOPIC OF THE KETONES IN THE HUMAN ORGANISM

In the following the ketone (acetone) question is dealt with somewhat in detail. Particular stress is laid upon the mode of production of the acetone bodies in the human organism and the rationale of the "yolk cure" in the suppression of these substances. Again, some material has been added that will facilitate the understanding of the mooted question in its broader bearings. Without a careful reading of these supplementary notes much that has been said in the previous chapters cannot be fully comprehended.

I

THE ACETONE BODIES IN THE URINE AND THE FERRIC CHLORID REACTION

It has become a matter of routine with many to employ the ferric chlorid reaction when examining diabetic urines and to depend upon it for the recognition of diabetic acidosis. Clinical reports nowadays found in American as well as in foreign literature, in which apparently sole reliance upon the ferric chlorid reaction for the

detection of diabetic acidosis has been placed, are becoming so numerous, that a word of caution as regards the insufficient mode of testing is certainly in order.

The acetone bodies — beta - oxybutyric acid, aceto-acetic acid (diacetic acid) and acetone—are interrelated, it is true, and are derived by successive processes of oxidation from their common progenitors: the caproic, valerianic and butyric acids. However, contrary to a general supposition, the three acetone bodies do not always co-exist and never occur in a definite ratio to each other. Acetone is the least important substance of this group, while beta-oxybutyric acid represents the most dangerous factor of acidosis. This at least is the opinion prevailing at the present day. As a matter of fact, only a small portion of the acetone occurs as such in the urine; the greater amount is yielded by the aceto-acetic acid (diacetic acid) on standing of the urine or when the reagents are added. In reality an acetonuria is therefore a diaceturia.

Aceto-acetic acid (diacetic acid) is the only acetone body giving the ferric chlorid reaction. While the reaction is obtained in most instances of diabetic acidosis, there is a certain proportion of cases in which it does not ensue, at least not on all occasions. When a diabetic urine is being accumulated for twenty-four hours for the glucose determination—a common and necessary practice—its diacetic acid may have become com-

pletely oxidized to acetone at the time of examination. To avoid this eventuality only freshly voided diabetic urines should be subjected to the ferric chlorid test. Diabetic urines which have been standing for a number of hours must be examined for acetone by one of the direct acetone tests, preferably by the sodium—nitroprussiate—potassium hydroxid—acetic acid—method (Legal's test).

Beta-oxybutyric acid, one of the paramount acid factors in diabetic acidosis, is not always found in the urine associated with the other acetone bodies. This is especially the case in far advanced instances of acid intoxication when the oxidative qualities of the diabetic organism are interfered with to such an extent that beta-oxybutyric acid virtually represents the last stage of the oxytransformation of aforementioned fatty acids of low molecular weight. Of course, the ferric chlorid test will not respond if beta-oxybutyric acid is the sole representative of the acetone group and the tests for acetone as such will likewise be negative. Inasmuch as we do not possess a simple reaction for the detection of beta-oxybutyric acid this is never searched for by the practitioner, and many a case of acidosis occurring at the time of examination without the lower homologues of oxybutyric acid simply remains unrecognized, and this at a period when vigorous treatment is especially indicated.

In pronounced instances of diabetic acidosis

when oxybutyric acid exists in large amounts in the urine, especially when it be associated with some butyric acid, its immediate progenitor—as is not infrequently the case—its presence can always be readily detected by its penetrating rancid odor.

To summarize:

1. A positive ferric chlorid reaction is usually indicative of a state of acidosis, but the reaction is solely due to the presence of aceto-acetic acid (diacetic acid).

2. A negative ferric chlorid reaction does not preclude the presence of either oxybutyric acid or acetone.

3. The ferric chlorid test should be applied to the freshly voided urine only, as the entire aceto-acetic acid or a portion of it, by a loss of CO_2 , may be converted into acetone if the urine be standing for some time.

4. As a rule, no acetone at all, or only a small amount of it exists in a preformed state in the freshly voided native urine. Standing of the urine or reagents causes liberation of acetone from aceto-acetic acid.

5. The presence of aceto-acetic acid or of aceto-acetic acid and acetone does not of necessity point to the occurrence in clinical amounts of beta-oxybutyric acid.

6. Oxybutyric acid is one of the paramount acid factors of diabetic acidosis so far as we know at the present day.

7. Oxybutyric acid which is not demonstrable by the simpler testing methods may be readily discerned by its rancid odor.

8. A direct search should invariably be made for acetone by one of the other acetone tests.

II

ARE THERE KETONES OF INTESTINAL PRODUCTION?

This chapter simply deals with the almost forgotten question:—Are some of the ketones of enterogenous formation? Acidosis, as such, is, therefore, only mentioned in a casual way.

In many cases of acidosis beta-oxybutyric acid is found in excessive amounts. However, it is by no means the only low fatty acid that contributes toward the acid intoxication. The other members of this series—proprionic, valeric, capric, enanthylic, caprylic, pelargonic and caproic acids are probably as important in the production of acidosis as are the members of the butyric acid group themselves. Furthermore, besides acetone (C_3H_6O), the ketone yielded by acetic or acetoacetic acid, the ketones formed from the successive members of the fatty acid series, differing from one another by twice CH_2 , undoubtedly participate in the production, or are concomitants of the clinical pictures that are erroneously ascribed to the acetone bodies or their direct progenitors. Such ketones are propione ($C_5H_{10}O$) yielded by

propionic acid, butyrone ($C_7H_{14}O$) from butyric acid, and valerone ($C_9H_{18}O$), a product of valeric acid. The close chemical relationship of the successive members of the fatty acid series and that of their respective ketones, and the facts that they are, to the greater part, volatile liquids which are readily intermiscible and are subject to the same chemical reactions, give strength to the assumption that one member of the series of fatty acids or ketones may preponderate in a given case, but that it is hardly probable that these single members are present to the exclusion of all the others. Originating in the organism from practically the same source or sources and being affected by the identical chemical influences, it is obvious why more than one of the lower fatty acids and more than one of their ketones are apt to occur at a time, and why the phenomena of acidosis, which are by no means invariable and uniform, must of necessity be the result of the conjoint activity, or be the concomitants of various fatty acids and various ketones.

It is probably true that there is no case of general acidosis in which acetone or its immediate forerunner, diacetic acid, cannot be demonstrated in the urine; this, however, is no conclusive proof that other ketones or their corresponding fatty acids are not found associated with the former. It is simply the readiness with which acetone and diacetic acid are detected in the urine that has given them a clinical prominence which, in reality,

they do not deserve. Were the other ketones and fatty acids as easy of demonstration, the acetone bodies and their direct progenitors would not almost exclusively be held responsible for the production of acidosis.

While the occurrence of acetonuria may, therefore, furnish convincing evidence of an imminent or already established acidosis, it is in itself no proof that other members of the low fatty acid series have not participated in establishing this abnormal state, or that a ketone or ketones other than acetone stands at the foundation of the anomaly.

The question concerning us, however, is not that of acidosis but of the presence or production of ketones prior to absorption.

Hilliger,¹ and with him the modern clinical school, is of the opinion that the liver is the place of formation of the acetone bodies. They assume that here they are probably generated as intermediary metabolic products which, physiologically, are almost entirely oxidized to CO_2 and HO_2 . Borchardt² had eight years previously maintained the same standpoint, namely, that the acetone bodies are intermediary metabolic substances, resulting when carbohydrates, glycosides and glycerine are withheld from oxidation.

There is little doubt, if any, that the bulk of the ketones that are produced after the stage of absorption are due to a certain form of hepatic insufficiency. That some of the ketones, however,

may reach the intestinal tract in a preformed or nearly preformed state, or that they may be yielded and elaborated during pancreatic and intestinal digestion will be evinced in the following lines.

Some recent writers, particularly Howland and Marriott³ maintain that hyperpnea virtually spells acidosis. This is undoubtedly true. When there is an intestinal ketone formation and the liver is functioning properly, a ketonemia will either not ensue at all or, it will be so transitory and insignificant an occurrence that a hyperpnea will not be produced. In other words, ketones in the intestinal tract may give rise to various intestinal affections without concomitant ketonemia and hyperpnea. In fact, the confinement of ketones within the intestines is in almost every instance not followed by any evidence of intoxication or, for that matter, may not even result in any local clinical symptoms.

However, many diarrheal disorders, preeminently in childhood, are caused by the irritating activity of ketones upon the intestinal mucosa. To understand the untoward local influence of most of the ketones I wish to recall the abundant experimental and clinical work anent the fatty diarrheas, so-called. Accordingly, in intestinal ketone formation we have to deal with a vicious circle, i.e., a perverse disintegration of fatty substances in the alimentary tract on account of pancreatic or intestinal insufficiency, a local hyper-

emia, catarrh, etc., and a continuous irritation of the local process for reason of the continued perverse or increased elaboration of ketones from the volatile fatty acids. This vicious circle is only broken by withholding the foodstuffs that yield measurable quantities of ketones.

A fatty diarrhea is to all intents and purposes a ketone diarrhea, that is to say, it is not produced by the fatty acids of low molecular weight but, in some degree at least, by the ketones they have yielded. The ketone diarrhea, like the fatty diarrhea, may ensue without any systemic symptoms whatever. If it continues for more than thirty-six hours it is usually accompanied by loss of strength and body weight. In case it prevails for a protracted period, either in a continuous or the more common intermittent form, it is liable to give rise to marasmus and general atrophy. Even in the gravest types of atrepsia it is unlikely that one has to primarily deal with an intoxication. Hyperpnea never supervenes in uncomplicated cases and is certainly not an evidence of enterogenously-formed ketones that have not penetrated the intestinal wall. In all these cases one is hardly justified in speaking either of a ketonemia or an acidosis. Ketonemia or acidosis may undoubtedly concur with enterogenous ketones; however, the clinical picture of intestinal ketonosis as such is due to the local action of the enterogenous ketones contained within the bowel.

Ketones, on the other hand, find their way very readily into the general blood stream. This is rather the normal occurrence when the liver does not sufficiently functionate to cause their physiological cleavage after they have been carried to the portal circulation. While in cases of acid intoxication the liver is the organ in and by which the acetone bodies are produced as intermediary metabolic substances, and are not physiologically split up into simpler bodies in intestinal ketonosis, when the ketones enter the alimentary tract either in a preformed state or are elaborated therein, the normally functioning liver is the principal line of defense against qualitatively or quantitatively abnormal intestinal substances.

Symptoms

The symptoms of intestinal ketonosis are in a degree negative in character, but positive evidence is not missing.

General Appearance.—The patient usually looks undernourished, weak and anemic.

Diarrhea.—Loose or watery discharges from the bowels are the rule. The reaction of the stools is always acid. These possess usually a butyric-valeric odor. When recently passed, acetone may be contained therein. (Acetone in the stools may be detected in the following manner: The fresh feces are first well diluted with water, acidified with acetic acid, and then distilled. The distillate (10 c.c.) is treated with a solution of iodine in ammonium iodid; this results in the

formation of iodoform and a black precipitate of nitrogen iodine. The latter gradually disappears on standing, thus rendering visible the iodoform. This test is reliable, as it excludes disturbing factors and sources of error like alcohol and aldehyde).

The stools may be macroscopically fatty, but this is not necessarily the case. Mucus is of rather frequent, blood of infrequent, occurrence. The diarrheal attacks may alternate with more or less protracted periods of constipation.

Ketonemia and Ketonuria.—Absorption of ketones may take place, but ketonemia and ketonuria, when present at all, are most always of very brief duration.

Hyperpnea.—The amount of ketones which may be present in the blood at any one time is never sufficient to give rise to a hyperpneic condition.

Ketones in the Alveolar Air.—The alveolar air is free, or almost so, from ketones. The ketone odor emanates from the alimentary tract and not, as in ketonemia, from the lungs.

Ketones in the Intestinal Gases.—Ketones may be expelled with the flatus. Their presence can be demonstrated clinically by the sense of smell only. Of course, they are admixed with the other gases of fermentation and putrefaction. Propione, butyrone, valerone and acetone are likely to occur in the flatus when their respective progenitors are found in the feces.

Fever.—Temperature elevation may or may not be present. It may occur on one day, but may be absent on the next. In adults the eventual increase hardly ever surpasses two deg. F. In children below four or five years of age the rectal temperature may reach 104 to 105 deg. F.

Pain.—Abdominal pain is not always present; it is hardly ever of a severe type. This pertains to children as well as to adults. A pressure point sensitiveness is often found about the cecal region. In small children a scorching pain about the rectum and perineum is frequently met with. It is due to the irritating volatile acids and their ketones excreted in the feces.

The Seat of Production of Intestinal Ketonosis

Normally the ketones are readily absorbed on account of their great volatility. At the bottom of intestinal ketonosis there must therefore stand either (1), an insufficiency of the lacteal system or (2), the possibility that in intestinal ketonosis the ketones are not yielded until the residual ingesta, containing the lower fatty acids, have reached the large bowel. A number of clinical observations have convinced me that a structurally or functionally diseased cecum, hindering the free absorption of water, is the frequent seat of the formation and retention of enterogenous ketones. A few of the pertaining observations may here find mention.

Observation I.—Patient of Dr. Lack of Brook-

lyn. Married woman, twenty-seven years old, one child. Very frail. Undernourished and anemic. Penetrating valero-butyric odor from mouth when holding breath. The odor is independent from intake of food. Feces contain acetone as well as fatty acids and soap needles for a long period (two months). Occasional ketonuria. Chief complaints: Extreme prostration; diarrhea.

Examination: Nothing was found of pathological import, except cecal regurgitation and evidence of Jacksonian bands.

Treatment.—Operation by Dr. Lack. Removal of appendix and Jacksonian membranes; cecopexy. Complete recovery from intestinal ketonosis.

Observation II.—Married woman, thirty-eight years old, one child. Rather thin, anemic. Rancid odor from mouth when holding breath. Feces contain valeric, butyric, aceto-acetic acid, acetone and large amounts of mucus. No ketonuria. Chief complaints: Restlessness; pain over course of transverse colon; diarrhea alternating with constipation; dryness in throat.

Examination: A cecum of great length and very movable was found.

Treatment.—Operation by Dr. Fischer. Removal of appendix. Cecopexy. Complete recovery from intestinal ketonosis.

Observation III.—Man, thirty-six years old. Present weight 142 pounds, but has lost over thirty pounds in five months. Aceto-valero-buty-

ric odor from mouth when holding breath. Odor is practically always present. No acetonuria. Feces contain fatty acids, soap needles and acetone. Chief complaints: Pain and soreness in left inguinal region; irritability; attacks of diarrhea.

Examination: By colonoscope no sign of disease of rectum and sigmoid discernible. Gurgling sounds in the cecum, pointing to atony of this part of the gut, are much in evidence.

Treatment.—Operation by Dr. Fischer. Removal of appendix. Cecopexy. Complete recovery from intestinal ketonosis.

I could recount a number of similar cases in which an intestinal ketonosis disappeared after the removal of the appendix, bands and adhesions about the cecal region and with or without cecopexy. In other cases, especially in nurslings and children, operative interference is not indicated. In these cases a rational change of diet is always followed by a suppression of the intestinal ketonosis.

Sources of Intestinal Ketonosis

The mother substances of intestinal ketones are the volatile fatty acids. These volatile fatty acids are contained in milk and they form a goodly percentage of the fat of cow's milk, of butter and of some other milk products. Volatile fatty acids are also yielded as cleavage products of other fatty substances, of proteids and carbohydrates;

however, the amounts thus obtained are very small, as a rule. It stands to reason that practically all the volatile fatty acids in bottle-fed infants are yielded by the milk-fats.* In normal adults on a mixed diet the amount of volatile fatty acids is rather insignificant, unless there be a distinct alimentary disorder. When put on a milk regimen or when an adult consumes large amounts of butter or certain cheeses, the volatile fatty acids will increase at once.

The entire secret, then, to overcome intestinal ketonosis in infants as well as in adults, consists in the withdrawal of the milk-fats and the substitution therefor of fats of higher melting point.

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III

CONCERNING THE SUPPRESSION OF THE ACETONE BODIES IN DIABETICS

In whatever manner the substances of the acetone group arise, they are a symptom of inanition indicating decline of the diabetic organism.

* Besides, a certain proportion of the fecal acidity is undoubtedly caused by the intestinal microorganisms whose number and activity are often pathologically increased in many of the alimentary disturbances of infantile and adult life.

Acetonuria in the diabetic occurs independently of the degree of the glycosuria—that is, its intensity is not reflected by a corresponding intensity of the latter. On the other hand, the degree of acetonuria—when all circumstances are duly considered—may afford a fair insight as to the extent of tissue waste. Furthermore, the presence of an excess of the acetone bodies may point to either an inability of certain organs to convey the introduced alkalies to the structures in need of it, or else, to the insufficient diffusion and ionization of the salts in the body liquids.

The formation of the acetone bodies will not ensue as long as one can manage that the diabetic remains in a fair state of nutrition; if this proves impossible in the course of time—an inevitable occurrence supervening sooner or later—the general deterioration should be retarded by all possible means. The waste of the diabetic organism, in fact, may be so gradual, that urinary acetone may not appear in excess of 0.25 gram in the twenty-four hours.

If acetonuria and diaceturia occur in a pronounced degree, attempts should be made, repeatedly if necessary, to suppress the further production of the acetone bodies, or, if these fail, to keep their formation at a minimum. By adding carbohydrates to the nourishment the acidosis may decrease for the time being. v. Noorden⁴ highly recommends his “Oatmeal cure” for this purpose, which is carried out in the following

manner: Patient ingests every day for a period of from one to two weeks, to the exclusion of everything else, a soup composed of oatmeal 250 grams, butter 250 grams to 300 grams, and some vegetable albumin 100 grams. A portion of this soup is partaken of, every two hours.

There is no doubt that the degree of acidosis is, as a general rule, temporarily reduced when carbohydrates are admitted to the diet. However, they should not be partaken of for longer than three or, at the utmost, four days, as otherwise the inevitably increased glucose output will make itself felt in a manner not to be misunderstood. A short time after the carbohydrates are discontinued, the acetone bodies are again excreted in similar amounts as before the admittance of the starchy matters to the nourishment. Thus, although carbohydrates may alternately be permitted and withdrawn for some weeks, nothing has been gained in the end. It is true, a fatal issue might have been averted, but whether this was imminent is a matter of mere speculation. On the other hand, the ingestion of carbohydrates, irrespective of the form in which they are taken, whether of oatmeal, or of potatoes, as suggested by Mossé,⁵ or of milk, may occasion vastly more injurious results than would have ensued had they not been added to the food.

Furthermore, the oatmeal cure is not only disgusting to the patient, but it does not uphold the claims of its author. Oatmeal, in my experi-

ence, is not any better tolerated by the diabetic exhibiting acetonuria, than other amyloid substances, as buckwheat, or rice, for instance. Again, the very large amount of butter, 250 grams to 300 grams daily, which is to be incorporated into the oatmeal gruel, contradicts the rationale of acidosis therapy. Half a pound of butter, or more, ingested daily, is certainly apt to yield large quantities of acetone bodies in the diabetic; as a matter of fact, I have seen a case, after restriction to the oatmeal-fat-albumin compound for some days, in which the intensity of the acetonuria had not only not diminished, but had been appreciably augmented.

In his oatmeal cure, v. Noorden (who himself has shown that the ingestion of more than 150 grams of unwashed, and of more than 180 grams or 200 grams of washed butter—by the washing process butter loses its low fatty acids—is liable to be followed by an increased production of acetone bodies) orders the addition of from 250 grams to 300 grams of butter. Does he really think that the 250 grams of oatgruel suppresses the already existing acidosis and prevents the yielding of acetone bodies by the over-supply of butter?

If we wish to cope successfully with acidosis without aggravating the diabetic condition we can neither add starches to the nourishment for any length of time nor can we in most cases in-

crease the amount of protein substances.* Therefore, nothing remains but to turn again to the fats, that is to such fatty ingesta, which, if at all, contain but small amounts of fatty acids of low molecular weight. According to the investigations of Leo Schwartz,⁶ the fatty acids which may be considered mother substances of acetone bodies are valerianic, butyric and caproic acids; fatty acids of high molecular weight, like palmitic, stearic and especially oleic acids, yield comparatively little acetone. Hence, butter and cream, are productive of largely increased acidosis, while lard, suet and particularly olive oil do not cause a very marked augmentation of the acetone bodies.

The clinician who is attending a case of grave diabetes complicated by pronounced acidosis, finds himself in a dilemma. Were he to prescribe a diet consisting of nothing else but small amounts of proteins and large quantities of lard, suet or olive oil, the patient would soon be tired of this regimen and his nutrition would not only be not improved but his digestive organs might become seriously disordered. Besides, and this should not be forgotten, the vegetable oils are as a rule not well tolerated if they are used in larger than the ordinary amounts, and lard (frequently

* The albuminous ingesta in grave cases of diabetes do not only augment the glucose output but are liable to putrefy in the alimentary canal whereby butyric and valerianic acids, the precursors of the acetone bodies, are formed.

not well borne in even moderate quantities) and beef suet can neither be relied upon for suppressing an already existing acidosis nor to improve the nutrition of the diabetic organism.

There is only one fatty article of food which I found well adapted to the needs of the diabetic organism during acidosis—this is the yolk of the hen's egg. I have very rarely seen it produce or increase the acetone substances, or call forth digestive or assimilative disturbances in advanced diabetes.* Partaken of in the raw or semi-raw condition, fresh yolks may be consumed in almost any quantity without calling forth satiation or fulness.

The rationale of the "yolk cure," which will be described in detail, I attribute, among others to the following four main factors:

1. Palmitin, stearin, and olein, the fat substances of the yolk, occasion but small amounts of acetone bodies; in combination in which these fats exist in the yolk, they yield no butyric acid, or hardly any.

2. The large amount of lecithin, supplied to the organism by the yolk, tends to the restoration of nerve force and the amelioration of the cachectic condition.

* The "idiosyncrasy" for eggs not infrequently met in even healthy persons seems to be evoked in the first instance by the white of the egg. None of the diabetics whom I had under my "yolk cure," nor a number of other undernourished persons in whose diet yolks preponderated, developed an idiosyncrasy for the latter.

3. The occurrence in the yolk of a diastatic ferment assisting in the conversion of amyloid substances introduced by proteids and allowed vegetables.

4. The properties of the yolk to stimulate the digestive secretions.

The first and second factors are too well understood to be again exploited on this occasion. Concerning the third, Mueller and Masuyama⁷ found in the hen's egg, more particularly in the yolk, a diastatic ferment which has the ability of forming erythrodextrin, then achroodextrin, and finally isomaltose. The converting power of the ferment is less pronounced and less acute than that of the salivary and pancreatic ferment, but it can diastatize as much as forty-five per cent. of the food during the day.

As regards the fourth factor, I wish to state that Ssoborow, a pupil of Pawlow, some years ago demonstrated the marked stimulating influence of the yolk of the hen's egg upon the gastric glands; he found that the amount of gastric juice, obtained after feeding yolks to dogs whose stomachs had been isolated, was greater than after ingestion of any other articles of food. Obesersky,⁸ confirming these observations from the clinical standpoint, concludes that the yolk of the hen's egg exerts a pronounced influence upon the hydrochloric acid glands of the gastric mucosa and that the yolk not only stimulates the stomach to greatly increased secretory activity, but that

its frequent ingestion activates the acid glands to such a degree that yolks may be employed as digestants in affections characterized by lack of hydrochloric acid. My own observations upon the stimulating influence of yolks on the digestive secretions substantiate the contentions of the Russian investigators.

The "yolk cure" consists in the ingestion of from ten to forty yolks a day, together with a small amount of proteids and the non-avoidable carbohydrates furnished by the latter and certain allowed quantities of green vegetables. As each yolk contains about 5 grams of fat representing more than 46 calories, it takes about 21 yolks to furnish 1,000 and about 32 yolks to make up 1,500 calories. A diabetic weighing 60 kilograms, ingesting 21 yolks a day, would, therefore, obtain in fat alone 16.6 calories per day and kilogram of body-weight; taking 32 yolks a day the average for each kilogram of his absolute weight would be 25 calories. Yolks sufficient to yield from 10 to 15 calories per day and kilogram of body-weight, however, are all that are necessary to be ingested in grave cases of diabetes complicated by acidosis.

The "yolk cure," besides suppressing the acetone bodies, causes systemic and nutritive improvement, and increase of body-weight, vigor, and resistance. It also stimulates body-growth in the diabetic child or adolescent.

The "yolk cure" supplies but very small

amounts of nitrogen. However, it is body-albumin sparing in a much higher degree than any other dietary regimen. Of course, the excreted nitrogen, while the patient is under the strict "yolk cure," exceeds that which has been ingested; the deficit, however, is so trifling that, practically, it does not need to be taken into account. This is especially the case when the yolk days alternate with protein-fat (yolk) days. Again, when the glycosuria is moderate, so that larger quantities of albumin, together with yolks, instead of suet or butter, may be partaken of, a nitrogen deficit may be averted altogether.

The technic of the "yolk cure" is quite simple. The diet on which the patient had been, is at once discontinued; this is best done by omitting the next meal. In the meantime the patient's intestinal tract is evacuated, after which a high saline enema should be administered. When the decline of the patient has been very rapid, and when the glycosuria and acetonuria are of a high degree of intensity, the exclusive "yolk cure" must be continued until general improvement has ensued. In other, less pronounced cases, it may suffice to have a "yolk day" twice or three times a week, while the regular protein-fat (yolk) regimen is pursued on the other days. In a small percentage of the cases larger amounts of carbohydrates may be permitted when the improvement has continued for some time. The additional carbohydrates should be given in the form

of green vegetables, and not in that of flour, beans, rice, or potatoes. In whatever manner the yolk diet is altered, the yolks should continue to displace all other kinds of fat, and a strict yolk day should be pursued at least once or twice a week for a protracted period.

A sample of a yolk menu is given in the following table:

	Number of yolks.	Calories yielded by yolks, approximately.	Total calories, approximately.
<i>Breakfast:</i>			
Cup of coffee, with two yolks.....	2	92	92
Three boiled yolks.....	3	138	138
<i>Early lunch:</i>			
Egg-nog (three yolks, 30 cc. whiskey)..	3	138	270
<i>Dinner:</i>			
Celery soup (five yolks).....	5	230	230
Spinach, 250 grams, eight yolks.....	8	368	443
Cup of coffee, with two yolks.....	2	92	92
<i>4 P.M.</i>			
180 cc. sugar-free red wine, one yolk....	1	46	125
<i>Supper:</i>			
One plate soup (soup stock), five yolks..	5	230	250
String beans, 90 grams, three yolks.....	3	138	150
Total.....	32	1,472	1,790

(The administration of alkalies, although the influence of the latter upon diabetic acidosis, as pointed out by me⁹ seems to be a mere local and limited one, is an innocent procedure, and may be pursued as an adjuvant to the antiacetone diet. It is possible that alkalitherapy for a time averts

the enterogenous production of acetone substances, but it is a mooted question whether alkalies alone, that is, when the diet under which acetone has been formed is continued, will bring about cessation of an already active acetone production.)

In order to avoid monotony, variety in soups, vegetables, flavoring, and seasoning substances should be planned. The following cooking recipes for yolk dishes will be found of service:

PLAIN YOLKS

Boil, shirr or poach the whole egg—only the yolks should be consumed.

COFFEE

Stir the yolks of two eggs in a cup, then add one cup of hot coffee stirring constantly.

WINE SOUP

Let 150 c.c. of red (sugar-free) wine and the same amount of water, a little cinnamon and one clove come to a boil. Remove from the fire and stir in the yolks of two or three eggs stirring constantly. Add saccharin, if desired.

SPINACH AND ALL GREEN VEGETABLES

Boil in salted water until very tender—drain—add pepper and mash to pulp or press through colander. Then add to each ounce of pulp one raw yolk and mix well.

SALAD DRESSING

Mix the yolks of six eggs (well beaten) with salt, pepper, one teaspoonful of onion juice and mustard. And juice of half a lemon.

CELERY AND CAULIFLOWER SOUP

Boil a stalk of celery (cut fine) in 250 c.c. of salted water until very tender—strain and add to the boiling liquid the well-beaten yolks of five eggs. Season to taste.

Cauliflower which has been scalded is put on to boil with 250 c.c. of salted water, when very tender the whole mixture is passed through a colander and returned to the fire, then the well-beaten yolks of five eggs are added. Season to taste.

SOUP STOCK

For each plate of soup a quarter pound of shin meat, a quarter pound bone, some celery, parsley, one onion and half of a leek are required. Set on the fire with cold water and boil slowly for five hours. Strain, pressing meat to a pulp so as to extract all the juice. Then add five yolks, well beaten.

EGG-NOG

Mix 30 c.c. whiskey with 90 c.c. water and shake or stir briskly with the yolks of two eggs—add nutmeg, cinnamon, or lemon.

All yolk dishes must contain salt in sufficient

amount; salt not only aids in the assimilation of the yolk constituents, but is absolutely essential for the proper conduct of the metabolic and osmotic processes. The patient, as a rule, takes readily to the yolk cure. I have some patients who, although not eliminating acetone bodies any longer, continue of their own volition two or three yolk days in the week, and substitute, as much as possible, yolks for other fatty ingesta on the protein-fat days.

Generally, obstipation does not supervene while the patient subsists on the "yolk cure." However, if it ensues, high enemas, to be followed by some saline cathartic or a good dose of castor oil, to be followed by enteroclysis with sodium chlorid solution, should be at once administered.

Following are the histories of some cases of diabetes, complicated by acetonuria and diaceturia, which had been under the strict and modified yolk diet:

CASE I.—J. R., forty-five years old, is a diabetic since his thirty-ninth year. Between March and May, 1903, his affection, which had been of a comparatively mild type for five years, began to assume a grave character. On February 22, 1903, while under the diet which he pursued for the past three years, and which contained 150 grams of carbohydrates per day, he weighed $147\frac{1}{2}$ pounds, and his urine exhibited neither glucose, acetone nor diacetic acid. On May 10, his weight had declined to $146\frac{3}{4}$ pounds, and his urine (the

amount of which had not been ascertained) contained 1.5 per cent. glucose, but the tests for acetone and diacetic acid showed neither. On May 25, while under a rigid meat-fat regime, he excreted 4 per cent., or 90 grams of glucose, but no acetone or diacetic acid. On June 8 the diurnal amount of excreted glucose had diminished somewhat, but there were traces of acetone, and the ferric chlorid reaction was fairly pronounced. His weight was $145\frac{1}{4}$ pounds. About June 25, he consulted Prof. v. Noorden, at Frankfort, who informed me that he had found 2.3 per cent. or 56 grams glucose, and 1.5 grams acetone in the twenty-four hours' urine, and that the ferric chlorid reaction, although faint, had occurred.

Upon his return, on September 13, he weighed 144 pounds; his urine contained large amounts of glucose and some acetone, but no diacetic acid. On September 23, he excreted 181.6 grams glucose, a small amount of acetone, but no diacetic acid. On September 25, after two days of a green vegetable and fat (butter) diet, as advised by v. Noorden, he excreted but 83.4 grams glucose, but over 2 grams acetone, and also some diacetic acid. On September 29, his weight had declined to $143\frac{1}{2}$ pounds, the glucose output to 54.97 grams, the amount of acetone had not materially changed, the ferric chlorid reaction was positive. Keeping the patient upon a very rigid diet (complete exclusion of carbohydrates, diminished amount of albumin, no egg albumin, only the

yolks), he weighed $145\frac{1}{4}$ pounds on October 5, and had excreted during the previous 24 hours about 43 grams glucose, and traces of acetone; diacetic acid was not detected. Withdrawing all fatty substances except the yolks of eggs, he weighed, on October 12, $148\frac{3}{4}$ pounds. The glucose output had diminished to 38.18 grams; acetone had entirely disappeared. Diacetic acid was not present.

Olive oil, added to the diet at a later day, was followed by a slight output of acetone; addition of butter, tried on various occasions, caused pronounced acetonuria. Beef fat, in the amounts ordinarily employed, remained without influence upon the acetone excretion. The patient weighed $152\frac{1}{2}$ pounds on November 30, and passed as little as 10 grams glucose on January 10, 1904. His urine remained entirely free from acetone until the end of February. Since this time, small amounts of acetone, as well as of diacetic acid, are found occasionally. His present weight is $149\frac{1}{2}$ pounds. Since the middle of October, 1903, his diet consists of meat and fish and their natural fat substances, of gelatin, green vegetables, almond cake, coffee, and some brandy. The daily caloric deficit is made up by the yolks of hen's eggs, of which he had as many as 14 per day. These are incorporated into gravies and vegetables, and added to the coffee and brandy.

CASE II.—Mrs. J. D., forty-four years old, who gave birth four times, three times to stillborn

children, consulted me first on April 16, 1903. The patient has been affected with diabetes for about two years, and had exhibited all the initiatory symptoms of the diabetic state. She has lost about 20 pounds in body-weight. Her present weight amounts to $103\frac{1}{4}$ pounds. The quantity of urine voided in the twenty-four hours was 2,800 c.c. It possessed the following features: Specific gravity, 1,033; reaction, 0.45 deg. acid; glucose, 8.35 per cent.=233.8 grams in 24 hours, acetone, 2.25 grams; ferric chlorid reaction very pronounced. Albumin and evidence of renal lesion could not be detected. Patient was put on the rigid yolk cure for three days, after which the yolk days were to alternate with protein-fat (yolk) days for about two weeks, after this two yolk days a week for a protracted period. On May 7, the diurnal urine amounted to not quite 2,000 c.c., and the glucose output to 71 grams. *The excretion of acetone bodies had ceased.* Patient felt strong, and weighed $104\frac{1}{4}$ pounds. On May 21, the twenty-four hours' urine amounted to 1,850 c.c. and the glucose to 67.155 grams. The acetone substances had not reappeared.

CASE III.—M. F., forty years old, born in Russia, butcher. Symptoms of diabetes first noticed in December, 1902. Highest weight, 155 pounds. Weight on May 2, 1903, $123\frac{1}{2}$ pounds. Urine on the latter day amounted to 5,400 c.c. and contained 336.96 grams glucose and large quantities of acetone and diacetic acid. Rigid

protein-fat (butter) diet reduced the diurnal urine to 3,200 c.c., and the glucose to 85.12 grams after two weeks. The reactions for acetone and diacetic acid had become more intense. Patient was put on the rigid "yolk cure" for two days, and on alternating yolk days and protein-yolk days for two weeks longer, with the result that the daily glucose output had diminished to less than 25 grams and that the *acetone substances had entirely vanished*.

CASE IV.—W. S., twenty-five years old, merchant, first consulted me on June 4, 1904. The diabetic phenomena were recognized about two and a half years ago. His highest weight had been 155 pounds; present weight 109 pounds. Patient is also affected with advanced pulmonary tuberculosis. The saliva contains considerable amounts of glucose. Urine, quantity voided at my office at one time 900 c.c., specific gravity 1,021, glucose 5 per cent., acetone considerable, diacetic acid ferric chlorid reaction positive. No evidence of kidney affection.

Antidiabetic regimen, in addition thereto about 500 c.c. of sweet cream daily. On June 11 the glucose had been reduced to 2.4 per cent.; acetone and diacetic acid occurred in rather larger quantities. On June 19 patient who had not adhered to the prescribed diet excreted again 5 per cent. glucose, but somewhat less acetone and diacetic acid. His weight had been reduced to 107½ pounds. Protein-yolk diet and every third day

a vegetable-yolk regimen were ordered. June 25 the patient feels improved. Urine on last vegetable day about 3,000 c.c.; on last protein-day 4,000 c.c. Glucose, vegetable day, 3.12 per cent.; protein day, 5.55 per cent. Specific gravity, vegetable day, 1,020.5; protein day, 1,021.5 Acetone, vegetable day, none; protein day, traces. Ferric chlorid reaction, vegetable day, negative; protein day, faint.

CASE V.—Mrs. B. C., seventy-two years old, mother of 9 children. The patient was first seen by me on June 18, 1904. Diabetic condition dates back for some years. In addition thereto she has all the evidences of contracted kidney. Her highest weight was 140 pounds; present weight amounts to 108¾ pounds. Twenty-four hours' urine, 2,000 c.c.; contains large quantities of albumin and renal débris, 0.83 per cent. glucose; acetone, and diacetic acid present. Vegetable yolk-milk diet (from 12 to 22 yolks a day) was ordered, with the result that on June 25, *glucose, acetone, and diacetic acid had completely disappeared*, and the amount of serum albumin was materially reduced. After addition of 100 grams of bread to the diet she was still free from glucose, acetone, and diacetic acid on September 1, when she weighed 110 pounds. She still subsists on the same diet, taking little meat, but about 12 or 15 yolks a day. The amount of serum albumin eliminated by the urine has been considerably reduced since treatment was started.

CASE VI.—G. H., fifteen and one-half years old, was referred to me on March 29, 1904. Eight months before he began to waste away. About three months before the family physician recognized diabetes. The diurnal urine amounted to 6,772 c.c.; the specific gravity was 1,032.5; glucose 5.5 per cent., that is, 372.46 grams; it also exhibited large amounts of acetone and diacetic acid. He weighed 99 pounds. A moderately rigid diet with 100 grams wheaten toast was prescribed. On April 9 the twenty-four hours' urine amounted to 3,500 c.c.; specific gravity, 1,029; glucose, 10 per cent. (350 grams). Acetone and diacetic acid continued to be excreted in undiminished quantities. The patient's weight had declined to 98½ pounds. Rigid proteid-fat (butter) diet was ordered. On April 15 the diurnal urine amounted to 3,000 c.c.; specific gravity, 1,024; glucose, 7.5 per cent. (225 grams). *The amount of acetone and diacetic acid was enormously increased.* A proteid-yolk diet, and twice a week a vegetable-yolk day, were ordered (23 yolks on the latter days).

On April 29, the urine, vegetable day, amounted to 1,750 c.c.; on the previous protein day it had amounted to 2,500 c.c. The urine from the vegetable day exhibited 2.5 per cent., that from the proteid day, 3.33 per cent. glucose. Acetone and diacetic acid, although eliminated in decidedly smaller quantities, still occurred to the amount of 1.75 grams on the last vegetable day, and to that of 2.25 grams on the last proteid day. The

patient's weight had risen to 107 pounds. The "yolk cure" was ordered to be continued.

On May 12, a vegetable day, the urine amounted to 1,500 c.c.; on the previous protein day its amount was 2,250 c.c. The urine of the vegetable day had a specific gravity of 1,020.5, contained 2.5 per cent. glucose, gave a faint ferric chlorid reaction, but did not exhibit acetone in more than the normal amounts.

The urine of the protein day possessed a density of 1,027.5, and contained 6.25 per cent. glucose. Acetone was present in considerable quantities and the ferric chlorid reaction was very pronounced. The patient weighed 111 pounds.

On May 26 the urine (vegetable day—28 yolks) amounted to 1,750 c.c.; had a specific gravity of 1,016, and contained *neither glucose, acetone, nor diacetic acid*.

The urine of the last protein yolk day (7 days) was the same in amount, had a specific gravity of 1,018, and contained 0.25 per cent. glucose *but no acetone or diacetic acid*. The weight had increased to 113 pounds. Diet continued as heretofore.

On June 13, a vegetable day on which 23 yolks were consumed, the urine amounted to 1,250 c.c. It had a specific gravity of 1,012, a general acidity of 0.3 deg., and contained *neither glucose, acetone, nor diacetic acid*.

The urine of the last protein day amounted to 2,600 c.c., had a specific gravity of 1,017, a general

acidity of 0.3 deg., and contained *neither glucose, acetone, nor diacetic acid*. The body-weight had been raised to 116¾ pounds. On the last few vegetable days patient consumed only parsley which had been cooked in ham or beef water. The yolks were incorporated with the parsley. Treatment continued as heretofore.

On July 6, a protein day, urine was excreted to the amount of 4,250 c.c. It exhibited a specific gravity of 1,019, but no glucose, acetone, nor diacetic acid.

The urine of the last vegetable-yolk day amounted to 2,125 c.c., with a specific gravity of 1,014. It contained no glucose, acetone nor diacetic acid. The weight had declined to 115 pounds. Treatment as heretofore.

On July 27, a vegetable-yolk day, on which patient had consumed 30 yolks, the renal secretion amounted to 2,000 c.c., had a specific gravity of 1,015.75, and contained no abnormal substance of any kind.

The urine of the last protein-yolk day amounted to 2,900 c.c., had a specific gravity of 1,019.25, and showed neither glucose, nor any of the acetone bodies. The body-weight had declined to 113½ pounds.

On August 17, a vegetable-yolk day (17 yolks), patient voided 1,100 c.c. urine, the density of which was 1,010.75. It exhibited no anomalous constituents.

On the last proteid day the urine amounted to

2,600 c.c., possessed a specific gravity of 1,020.5, and contained no abnormal substances, nor normal constituents in excess. The discrepancy in discharged solids is very pronounced, viz., 22 grams on the vegetable day, 116.5 grams on the protein day. Patient yawns incessantly, and weighs 116½ pounds.

On September 14, a vegetable day, the urine amounted to 1,500 c.c., and had a density of 1,018; it contained no abnormal substances.

The urine of the last protein-yolk day amounted to 2,500 c.c. It exhibited a specific gravity of 1,019, but no glucose or acetone bodies. The patient is growing fast, feels very well and strong and consumes from 20 to 25 yolks on the vegetable days and from 9 to 12 yolks on the proteid days. His weight was 118 pounds.

On October 15, a protein day, the urine was excreted to the amount of 2,250 c.c.; its density was 1,021. No glucose or any of the acetone substances were contained in it.

The amount of the urine of the last vegetable-yolk day was 1,500 c.c.; its specific gravity 1,020.75. No abnormal substances were detected in it.

The patient feels very well and has been working in a factory for the last two weeks. He consumes from 5 to 9 yolks on the protein days, and from 22 to 30 yolks on the vegetable days. He continued to grow. His weight was 122½, and later on 126½ pounds.

A study of the cases dwelled upon in the foregoing evinces the indisputable value of the "yolk cure" in ameliorating the diabetic condition and in suppressing the production of the acetone substances. While I do not believe in dogmatic dietary rules, I maintain that the principles underlying the "yolk cure" are rational in combating diabetes complicated with excessive production of the acetone bodies.

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IV

THE "YOLK CURE" IN THE TREATMENT OF THE
UNDERFED

Who is underfed?—Some time ago I attempted to answer this question.¹⁰ I concluded that underweight and underfed are *not* synonymous conceptions, that an individual of underweight but in metabolic equilibrium is not underfed, and that in the real underfed there is always a traceable disease or disorder which, directly or mediately, prevents normal ingestion or complete utilization

of foodstuffs, giving rise thereby to bodily decline.

Does overfeeding alone avert the further deterioration or does it tend to restore lost body-substance in these instances? No,—overfeeding, on the contrary, may even call forth aggravation of the patient's condition on account of overloading, overburdening the digestive organs and overproduction of alimentary poisons.

As overfeeding *per se* is not a rational therapeutic procedure, what ought we to do to prevent further waste and to cause reparation of lost body-substance? We must attempt to prepare the organism so that the nutrient material is received properly, and that it is elaborated and assimilated.

It is absolutely useless to try to check bodily decline by forced feeding, without paying attention to the condition of the digestive organs and the general assimilatory qualities. As soon as the organism is able physiologically to dispose of the ingesta the tide of body-decay is stemmed in all those instances in which some digestive-assimilative disturbance, directly or remotely, stood at the foundation of the malnutrition. In advanced cachectic states, of course, when reparation of the digestive and assimilative functions cannot be any longer accomplished, body-decline will continue, uninterruptedly, as a rule.

We are wont to consider a number of chronic affections as “wasting diseases.” Such patho-

logical states, however, occasion little or no body-waste so long as the digestive and assimilative properties are maintained at or near the physiological standards. In chronic ulcerative phthisis, for instance, it is often astonishing how little loss of body-substance has occurred, even in cases which have very far progressed. In such cases, the inroads made by the disease itself are in a measure counteracted by the good digestion and assimilation.

The treatment of the so-called wasting diseases resolves itself in the prevention of waste. The secret of success in the treatment of consumptive affections lies in the proper management of the organs of digestion and assimilation. The most potent factor in maintaining physiological digestion and assimilation is an adequate food-supply, that is, a nourishment not only sufficient as regards the quantity, but one which the declining organism with the weakened or impaired organs is able to anabolize, one, in other words, which furnishes the needs of the specifically affected and altered organism. If the food is not adapted to the digestive and assimilative capacity of the patient, he will slowly starve, although his food-receiving organs may be filled with it to the point of bursting.

As a matter of course, measures other than dietary fulfil a certain, more or less important, purpose in the management of the condition of subalimentation; their specific value, however, is

mostly of a negative, of a defensive character so to speak, as their application does not tend to actually overcome body-decline and much less to increase body-weight, but to a retardation of metabolic and catabolic processes.

The most rational therapy for the digestive and assimilatory apparatus of the underfed as well as for the condition of underfed, is the one in which the diet plays the most prominent rôle, that is that diet which is not selected for the disease but for the individual, that diet which is altered as the patient's condition alters, that diet, in short, which does not call forth anorexia, sitophobia, cardiac irritability, dyspnea, or other untoward phenomena, but that diet which is the least bulky, the least burdensome to the alimentary organs, the richest in heat-units, and the most potent protector of body substance.

In the "yolk cure" we have a dietary regimen exhibiting all the advantages of a nutriment of the first order without its usual drawbacks.

Under "yolk cure" I understand a dietary regimen in which the greater portion of calories is yielded by the yolk of the hen's egg, and in which the latter forms the only fatty substance. Besides this *rigid* "yolk cure" we may speak of *modified* forms of the same. A modified "yolk cure" is a dietary regimen which is either (a) not a succession of "yolk days" (that is when days on which yolks do not preponderate in the diet are inserted), or (b) when the yolks al-

though contained in the diet in a certain amount, do not furnish the bulk of the calories and are not the sole representatives therein of the fatty ingesta. In a majority of instances a modified "yolk cure" will be found to offer certain advantages over the strict regimen when the patient's alimentary tract is in good condition and after subsidence of the acute decline, or after the patient has started to gain in body-weight. Moreover, the "yolk cure," modified according to the individual needs and desires and remodified from time to time in accordance with the changed conditions in the organism is the only means by which a yolk diet may be continued for many months and even for years.

The yolk of the hen's egg in the raw or half-raw state is very readily digested. Experiments conducted by me in the healthy human stomach show that two raw yolks (slightly seasoned) leave the stomach in 70 to 90 minutes; three in 70 to 100 minutes; four, in 80 to 100 minutes. One yolk taken in hot water and whiskey leaves the stomach in 50 to 70 minutes; two leave the stomach in 50 to 75 minutes. One yolk taken in a cup of hot coffee (some sugar, or milk) leaves the stomach in 60 minutes; two leave the stomach in 60 to 70 minutes.

The "idiosyncrasy" for eggs which undoubtedly exists in a number of even healthy individuals, in my opinion is solely due to the white of the egg. I have never found it when the yolks only

were partaken of. The white of the egg, containing the bulk of the latter's protein substances, in the face of retarded digestion, in gastric motor insufficiency or in any other condition detaining the ingesta in the gastrointestinal canal, is liable to yield hydrogen sulphid and ammonia; the yolk which is at no time lodged in the stomach as long as the egg white, does not exhibit the elements for the production of hydrogen sulphid to the degree that the latter could call forth any untoward consequences.

Yolks are very well assimilated. This was proved by the comparatively small amount of fat in the feces when yolks had formed the exclusive fatty substance in the diet. It is a known fact that the higher the melting point of a fat the slower and more incomplete will be its assimilation. The raw yolk, beaten up, being liquid, is quickly and almost totally absorbed. According to my own findings from 1.5 to 3.5 per cent. of the yolk-fat reappears in the feces. The great absorbability of yolk-fat will be appreciated when we understand that milk-fat reappears in the feces of the adult in the amount of 4.4 to 6.6 per cent., and in those of the nursling in that of 5.1 to 7 per cent. The residue left by yolk-fat in the feces is smaller than that of any other animal fat.

Yolks are well-borne and well-liked in almost any amount by almost every individual. This cannot be said of any of the other fatty ingesta. Suet, lard, and tallow are not well tolerated by

the ordinary stomach in large amounts and certainly not in quantities that yield the bulk of the necessary heat-units. A great proportion of these fats, 10, 12, and more per cent., usually reappears in the feces. Olive oil, like all vegetable fats, is not well-borne in larger than the ordinary amounts. Besides, the individual of our zone does not easily develop a taste for large quantities of vegetable oil; the acquisition of an aversion for it is much more frequently the case. Cream and butter are agreeable to the patient, as a rule, but when taken in amounts to furnish the mass of body-fuel they are liable to decompose in the stomach, setting free thereby fatty acids of low molecular weight. These low fatty acids tend to derange the digestive functions and the systemic equilibrium. Moreover, they are the forerunners of the so-called acetone bodies which, with more or less justification, have been brought into casual relationship to the coma of the diabetic and various other, apparently autotoxic, conditions.

Furthermore, the yolk of the hen's egg, as has already been shown, contains a diastatic ferment assisting in the conversion of amyloid substances. It does not exhibit the fermentative qualities of either the pancreatic or salivary starch-converting enzyme; its ferment-properties, however, are important nevertheless and should not be underestimated. Again, yolks are efficient stimulators of gastric secretion and may be looked upon in

certain respects as digestants. They may be par-taken of in hydrochlorhydria and in all conditions characterized by deficient flow of the gastric juice.

That lecithin is contained in the yolks is a fact too well known again to receive attention on this occasion. The ingestion of yolks hence may tend to the restoration of nerve force and the amelioration of the state of subalimentation.

An average yolk of the hen's egg contains between five and six grams of fat, representing about fifty calories. These, as we have seen, are almost totally assimilable calories. It takes, therefor, about twenty yolks to furnish one thousand and about thirty yolks to make up fifteen hundred calories. From eight to twenty calories, yielded by yolks, per day and kilogram of body-weight, will be found necessary in overcoming the condition of the underfed.

Yolks supply but very small amounts of nitrogen. However, it is body-albumin-sparing in a much higher degree than any other article of food with which I have experimented. Of course, the excreted nitrogen while under the rigid, unmodified "yolk cure" exceeds that which had been ingested; the deficit, however, is so trifling that, practically, it does not need to be taken into account. The modified "yolk cure," on the other hand, may be so devised that the patient remains in nitrogen balance.

In the preceding chapter "Concerning the Suppression of the Acetone Bodies in Diabetics," I

have already referred to the "yolk cure" as it should be pursued in diabetes complicated by acetonuria. I have reported some cases in which after adherence to the specific regimen the excretion of acetone substances had completely ceased and in which the glycosuria, at the same time, had diminished or vanished altogether. In all these cases, the "yolk cure" had called forth systemic and nutritive improvement, and increase of body-weight, vigor, and resistance. In one of the reported instances, that of a diabetic boy, $15\frac{1}{2}$ years old, whom I presented to the New York State Medical Association, excreting very large amounts of sugar and acetone substances, the body-weight increased, while under the "yolk cure" from $98\frac{1}{2}$ pounds to $126\frac{1}{2}$ pounds.

While in the strict antidiabetic regimen carbohydrates as well as proteins must of necessity be partaken of in more or less restricted amounts, the "yolk cure" for the non-diabetic underfed does not take the question of carbohydrates and proteins into consideration. Quantity and quality of each foodstuff, however, have to be determined when treatment of the non-diabetic underfed is undertaken. We should ascertain whether, when, and in what amounts a certain type of food is tolerated by the stomach, to what extent the nutritive substances are elaborated during the process of digestion, and what proportion of the nutritive and calorific principles is utilized for the general processes of anabolism. This is by no

means an easy task in the majority of cases of underalimentation; a minute study of the individual digestive, elaborative, and assimilative conditions, however, is imperative if we wish to augment absolute and specific weight of the underfed individual. Simple clinical methods at the disposal of every practitioner enable him to study his patient's alimentary qualities to the extent that he can prescribe the indicated dietary regimen in many of the common forms of malnutrition. In other, rarer, cases in which clinical observation and simple diagnostic methods alone do not afford sufficient insight into the alimentary conditions, resort must be taken to more complicated methods of examination and determination. The latter, not easy of execution in many instances, are but rarely performed by the general practitioner.

One fact we may be assured of, namely, that the yolks are well-tolerated by almost every underfed individual. Yolks hence are to be the basis of the nourishment of the underfed. It is for the clinician to determine in what kind and in how much of a menstruum the yolks are to be ingested. Here it is where the difficulty arises. In some cases it will be found the yolks are best administered in milk, coffee, or tea. In others in the form of a modified egg-nog. Their beneficial effect in many instances are only noticeable when they are taken together with certain carbohydrates in suitable amounts. Taken in soup or

broths, together with beef, lamb, or chicken, they often give rise to bodily improvement which does not ensue in the same degree when other types of ingesta serve as vehicles. Again, in other cases, especially in those of pulmonary tuberculosis, an ordinary mixed diet in which the fat substances are merely replaced by the yolks, is frequently all that is necessary to stimulate the assimilative properties and to cause increase of weight, vigor, and resistance.

If a patient who is not in the last stages of a consumptive affection, does not gain in body-weight while he is under the "yolk cure," the latter as a rule is not properly executed, that is, the food which is partaken of together with the yolks is either not the right kind or is ingested in amounts not suited to the alimentary condition. While the proper kind of nutriment favors the specific yolk action, food wrong in character or amount, or in both, retards or suppresses the absorption of the yolk constituents.

On the other hand, the yolks facilitate the digestion of certain amounts of carbohydrates (diastatic ferment) and that of comparatively large quantities of proteins (stimulation of gastric secretion). They do not seem to possess properties which render other fats added to the food easy of absorption. Again, impaired fat absorption appears to be directly responsible for the underfed state in a rather large percentage of cases. As experience (fecal examination) has

shown me that yolks are almost as completely absorbed in most instances of disease followed by loss of body-weight (in not too-far advanced stages) as they are in the normal individual, all fatty substances in the food should be replaced as completely as possible by yolks. When improvement has ensued, it might not be necessary to insist upon yolks as the sole fatty nutrients in all instances; some other fats, in limited amounts, may be added to the diet or what seems more rational, one or more days on which the yolks do not form an important part in the diet but on which the common fat substances are ingested in limited amounts, may be intercalated every week. On the other days, however, the yolks should continue to furnish the bulk of the heat-units or at least they should form the sole fatty matter entering the organism with the diet.

A sample of a yolk menu destined for a consumptive weighing 110 pounds (50 kilograms) whose normal weight ought to be 140 pounds (63.63 kilograms) but whose alimentary system admits the ingestion of some carbohydrates and of almost normal amounts of proteins, is given in the following. This patient should obtain food to the value of 35 calories per day and kilogram of body-weight, that is 1,750 calories in the twenty-four hours.

	Number of yolks.	Calories yielded by yolks, approx- imately.	Total calories approx- imately.
Breakfast—250 c.c. skim milk with 4 yolks	4	200	290
30 grams wheaten toast.....			75
Early lunch—Cup of coffee, 2 yolks.....	2	100	100
Dinner—One plate of soup, 4 yolks.....	4	200	225
Beef, very lean, 150 grams.....			125
30 grams wheaten toast.....			75
4 o'clock—250 c.c. skim milk, 30 c.c. whis- key, 3 yolks	3	150	370
Supper—Porridge of farina or rice, 100 grams, 1 yolk, skim milk.....	1	50	350
Apple sauce, 75 grams.....			30
At bedtime—Night cap (90 c.c. hot water, 10 c.c. whiskey, 1 yolk, teaspoonful granulated sugar)	1	50	110
Total	15	750	1,750

In a prolonged yolk diet, the proportion of the various types of nutritives may and must be altered according to the prevailing alimentary circumstances; a dietary as outlined in the foregoing, however, may be looked upon as a standard in the suitable cases. The whole eggs may be well tolerated in certain cases. When whole eggs are employed extra yolks may be added to them. In order to avoid monotony and aversion, the latter being invariably the consequence of the long-continued use of the same kind of meat or of the same kind of farinaceous material, a variety of dishes in which yolks may be incorporated should be devised.

All yolk dishes must contain salt in sufficient amounts. Flavoring and seasoning substances

may be added to the various articles of food as long as there is no contraindication to their employment; it should always be borne in mind that spices are not inert substances and that their use may as well retard as increase digestive activity, as the case may be. As a general rule, patients under the "yolk cure" are not subject to constipation. Mild laxatives may be employed in cases tending to this condition.

The "yolk cure" in its various modifications has nothing in common with the legion of unnatural and irrational feeding systems extolled here, there, and everywhere. It is not a fad with me and I hope it will not become one with others. It has its limitations, to be sure, but if conscientiously employed in certain forms of malnutrition, it will increase the body-weight and restore bodily resistance when other dietary régimes have proved decided failures.

REFERENCE

- ¹⁰ Who is Underfed? Medical Record, May 21, 1904.

V

THE FAT QUESTION IN ITS RELATION TO THE
PRODUCTION AND CURE OF INFANTILE
MARASMUS

It is, of course, well understood that infantile marasmus, like any other condition of underassimilation, is not the consequence of the self-same primary cause in every instance. As a matter of fact, however, food physically, chemically or biologically unsuited to the needs of the delicate infantile organism is the most frequent instigator of the gastro-intestinal disturbances, which again are the usual forerunners of *athrepsia infantum*.

In breast-fed infants, pronounced marasmic conditions are of comparatively infrequent occurrence; in the overwhelming majority of instances, wasting and atrophy supervene in bottle-fed, that is, in artificially nourished, children. Milk modifications and substitutes, purporting to replace the physiologic nutriment, are numerous; some are fairly rational, but a majority *ad nauseam* to the earnest seeker of truth and still more so to the helpless babe, have sprung into the field in the last twenty-five years.

Frequently, no doubt, the onset of *athrepsia infantum* has been averted by one of the more rational milk modifications or substitutes; but this peculiar state of undernutrition is met with by the clinician of today in at least the same

proportional numbers as was seen by his predecessor twenty and thirty years ago. Furthermore, when infantile atrophy has once ensued, none of all the milk modifications or food preparations so far devised, and for that matter not even good breast milk, seems to possess any special virtues in checking the wasting.

I have no intention whatsoever to thresh over old straw, and I am loath to sit in judgment on the multitude of formulas for the attenuation and modification of cow's milk or the various methods of artificial infant feeding. However, I wish to dwell on a much neglected point in infant feeding, a factor which may be at the foundation of many an instance of *athrepsia infantum*. This factor is the *chemical* character of the fatty substances contained in the baby's nutriment.

While the quantity of the fat aliment has found frequent practical consideration (Biedert's Cream Mixture, Gärtner's Fat Milk, etc.), its quality—apart from various minor attempts at modifying the fat of cow's milk which, physically and chemically, differs widely from that of mother's milk—has hardly ever been taken into account when devising a food for the healthy and particularly for the diseased and undernourished infant.

The fat of mother's milk, of course, is and should be the physiologic fat aliment of the normal nursling; properly constituted and in amounts suitable to the needs of the individual infantile organism, it does not yield low fatty

acids to the extent of calling forth intestinal or other disturbance and is absorbed and anabolized in quantities guaranteeing normal growth and development. Production and secretion of the fat substances in human milk, however, occur not always in the same ratio, and their composition may vary considerably. As long as quantity and quality of the fatty components of mother's milk fluctuate between physiologic limits, normal fat decomposition and assimilation will not be interfered with, and unless there exists some anomaly of the other milk constituents, or a bodily insufficiency or a pathologic condition, the development of the baby—although progressing but slowly at times—continues uninterruptedly as a general rule.

On the other hand, if for protracted periods the fat substances of mother's milk occur in distinctly abnormal amounts, or in a less perfect emulsion, or if they are perverse in admixture or composition, gastro-intestinal disturbances, accompanied by severe metabolic irregularities or by toxic phenomena and terminating in bodily decline and atrophy, will supervene sooner or later. If permitted to run for any length of time, this wasting condition cannot always be checked and a fatal termination be averted, in instances even where the fatty principles of the mother's milk have meanwhile been excreted in normal amounts and composition, or in which the child has been fed on the milk of another woman.

Happily, extreme alterations in the amount and especially in the composition of the fat of the milk of a healthy woman do not ensue very often, and therefore a pronounced marasmic condition obtains comparatively rarely in the (at birth normal) breast-fed child.

In artificial nutrition, no matter whether a modification of cow's milk or a food preparation is utilized, the entire fatty principles are, as a rule, derived from the cream of cow's milk.

The fat of cow's milk, it is true, may be added to the food mixture in any desired amount; its composition, however, is liable to vary to a far greater extent than that of the fat of human milk, it being dependent on the breed and race of the animal, its individual characteristics, its age, how often it has calved, the time of lactation, the hour of milking, the season, quantity and quality of food, etc. Besides, and this is a fact but insufficiently expounded, the composition of the fat of cow's milk is at variance with that of the fat of human milk, differing especially in its far greater contents of volatile fatty acids, among which butyric acid is the most important. In the following table the discrepancies in the constitutions and proportion of the fats of mother's and cow's milk are quickly recognized:

PHYSICAL CHARACTERS

	Fat of Mother's Milk.	Fat of Cow's Milk.
Specific gravity at 15 deg. C.	0.966	0.949 - 0.946
Melting point.....	30 - 34 deg. C.	31.1 - 34.66 deg. C.
Solidifying point.....	19 - 22.5 deg. C.	35 - 38 deg. C.

CHARACTER AND PROPORTION OF FATTY ACIDS

Acid butyric, $C_4H_8O_2$	1.4	}	5.45	}	8.35
“ caproic, $C_6H_{12}O_2$			2.09		
“ caprylic, $C_8H_{16}O_2$			0.49		
“ capric, $C_{10}H_{20}O_2$			0.32		
“ lauric, $C_{12}H_{24}O_2$	49.2	}	2.57	}	53.90
“ myristic, $C_{14}H_{28}O_2$			9.89		
“ palmitic, $C_{16}H_{32}O_2$			38.61		
“ stearic, $C_{18}H_{36}O_2$			1.83		
“ dioxystearic, $C_{18}H_{36}O_4$	49.4	}	1.04	}	
“ oleic, $C_{18}H_{34}O_2$			32.5		

Very small amount of volatile fatty acids; oleic acid forms one-half of the non-volatile acids; of the solid fats, myristic and palmitic acids occur in larger amounts than stearic acid.

Volatile acids according to other analyses about 10 per cent. ; among the solid acids palmitic and stearic acids preponderate.

If we remember that mother's milk usually exhibits a somewhat larger percentage of fat substances than cow's milk—on the average 3.85 per cent. in the former and 3 per cent. in the latter—and that in order to compensate for the apparent deficiency, hand-fed children frequently obtain the fatty elements of cow's milk in larger proportion than they exist in the native product, we understand that the absolute discrepancy of the fat as it is given to the infant is even greater than its relative discrepancy as pointed out in the table. An extra amount of fat of cow's milk added to the child's food enhances the latter's contents of volatile fatty acids. Thus, in practice, the discrepancy in the proportion of the

fatty substances may even become a greater one.*

It is evident that a fat-compound consisting of 10 per cent. of volatile acids cannot be a rational substitute for a fatty nutrient into the composition of which these volatile acids enter in but comparatively small amounts. The most important of these volatile fatty acids is butyric acid, whose very presence in the fat of human milk has been denied by some investigators.

At any rate, the proportion in which it normally occurs in the fat of mother's milk is so small that any untoward action on its part has to be precluded. When it is exhibited in proportionally larger amounts for any length of time—a rare but always pathologic event—the human milk in which it is contained sooner or later ceases to contribute to the welfare of the nursling.

Inasmuch as the fat of cow's milk always displays a large quantity of butyric acid, and as this is the mother substance of the acetone bodies, the former would *a priori* not represent the ideal type of fatty nutriment for the infant. Besides this, the infantile organism is not particularly well adapted for the fat compound derived from cow's milk; it being unable to cope successfully

* It must also not be lost sight of that in human milk the fat is in a much finer state of emulsification than in cow's milk, which facilitates its direct absorption. This is of great importance, as there are in the infantile organism but insufficient amounts of biliary acids, and pancreatic development and function are as yet not far progressed.

with it even in a mere physical sense. This is evinced by the smaller absorption of the fat of cow's milk. Uffelmann¹¹ determined that the absolute amount of fecal fat after the use of mother's milk was normally about half that when cow's milk had been the nutriment (in the dried feces 0.8-0.9 gram fat *per diem* after cow's milk, 0.44 gram after mother's milk). A larger excretion of fat of cow's milk than of human milk, although not in as pronounced a disproportion as in the normal state, occurs also in pathologic conditions of the digestive tract.

The occurrence in the feces of absolutely and relatively larger amounts of fat of cow's milk is *prima facie* evidence of its more incomplete utilization by the youthful organism. As a matter of fact, however, the fat of mother's milk is also incompletely absorbed, the fat contents of dry feces when mother's milk is the habitual food fluctuating between 10 and 20 per cent. The younger the infant the larger the amount of non-absorbed fat. The dried feces of infants during the first week of life, according to Blauberger (Die Fæces, Schmidt und Strasburger, 1903), contain 40 per cent. of fat when mother's milk, and fully 50 per cent. when cow's milk has been ingested. The fecal fat, surplus fat, as it has been called by some, which is found, though in widely varying quantities, in all conditions possibly has to fulfil a certain mission. Maybe it is essential for a better evacuation of the infants' bowels, maybe

it acts as a protector of the intestinal mucosa, maybe its purpose is a different one altogether. This much we may put down as an irrefutable fact: The more the fat output by the feces approaches the minimum figure for each period of infant life—provided normal amounts of food exhibiting a proper ratio of fats have been ingested—the better the gastro-intestinal organs perform their work, the healthier the youthful organism, the brighter the outlook for its normal development.

However, I wish to dwell on the character and not on the amount of the fatty substances. The volatile, soluble fatty acids, those which are more or less soluble in hot water whereby their molecular weight diminishes and which include those members of the fatty acid series up to capric acid, occur, as we have seen, in unusually large amounts in the fat of cow's milk. Butyric, caproic, caprylic and capric acids are contained in the fat of cow's milk in from six to eight times the quantity in which they are present in that of human milk. As the physical and chemical properties of the milk-fat are dependent upon the absolute and relative amount of lower and higher and uncombined fatty acids, it is evident that such a vast discrepancy as that existing between the constitution of cow's milk-fat and mother's milk-fat, cannot be overcome by any possible modification of the former.

Milk fat, chemically speaking, is a compound

of mixed glycerin esters and not a simple mixture of triglycerides. According to Volhard¹² a certain degree of decomposition of the neutral milk fat into glycerin and fatty acid occurs already in the stomach. The neutral fats very likely undergo hydrolytic decomposition and are rendered soluble partly by saponification and partly (as fatty acids) by the action of the bile. Inasmuch as almost the entire fat contained in the thoracic duct occurs as neutral fat in the form of a fine emulsion, while acids and soaps are present in minute quantities only, we are forced to conclude that a reconversion into neutral fats must take place in the intestinal wall. The fat in the intestinal tract of the ingested volatile fatty acids is probably similar to that of oleic acid and the higher members of the fatty acid series. Nothing definite, however, is known in this respect. The volatile fatty acids appearing in the feces do not offer any information in this regard, as they are practically always the result of carbohydrate fermentation in the intestines; likewise, no adequate explanation is afforded by the urinary volatile fatty acids, as oxybutyric acid and its concomitants and derivatives which, in the opinion of some of the more recent investigators, are products of an incomplete oxidation of the fatty acids of higher molecular weight. That this is not always the case, even not when these acids appear in pathologic amounts, I think I have conclusively shown by the dieto-therapeutic test. (See

Chapter III of Part II, "The Suppression of Acetone Bodies in Diabetics.")

Acetone of supposedly intestinal formation has not infrequently been accused of being the promoter of periodical vomiting in children, of infantile eclampsia and of numerous other affections to which the young child is prone. Be this as it may, of one fact we may be certain, that butyric acid in quantities sufficient to furnish large amounts of acetone substances cannot have been yielded by carbohydrates or proteins, but must have been ingested as such. What is nearer than to assume that the fat of cow's milk, which contains abundant butyric acid, is the real instigator of a number of pathologic affections which with little justification have been ascribed to an autotoxic origin.

Turning from the acetone question which, in its relation to various infantile disturbances, offers a field of fertile discussion, and even more of vast speculation, we find that the volatile fatty acids as furnished by the fat of cow's milk are very decided irritants of the delicate intestinal mucosa of the infant. The ingestion of these acids is therefore the primary cause of many instances of gastro-intestinal irritation and disease followed by undernutrition, bodily retrogression and *athrepsia infantum*.

Gastro-intestinal irritation once set up, progresses rapidly when the nutriment is not changed, that is, in those instances, when the sup-

ply of fat is not materially altered. The alteration in the fat supply as exercised today being almost without exception a quantitative one, consisting of reduction, suspension and even increased supply of fat aliment—it is obvious that an improvement in the little patient's local and general condition, when the fatty constituents of the nourishment are at the foundation of the disturbance, is always more or less a matter of luck.

No doubt the proteins and carbohydrates of cow's milk or artificial foodstuffs quantitatively and to a greater degree qualitatively, also stand frequently at the bottom of the gastro-intestinal disturbance, which, of course, gives occasion to the development of infantile atrophy.* Their eventual unsuitability, however, is a matter of general information, while the inadequacy of the fat of cow's milk in the feeding of babes is known to comparatively few clinicians. It is true, they often withhold the milk totally in certain affections of the gastro-intestinal tract, to which fact the speedy recovery of the infant is undoubtedly to be ascribed, yet they are not aware that, in the given instance, neither proteids nor carbohydrates, nor eventual superabundance of bacteria, but the fatty constituents of the milk furnished the source of the pathologic condition or prevented its amelioration.

* It is not improbable that the greater amount of citric acid and inorganic salts contained in cow's milk exert an unfavorable influence upon the digestive tract, the osmotic conditions and the nutrition of the infantile organism.

While withdrawal of milk-fat in hand-fed infants may frequently result in cessation of the local disturbance, it is obvious that the infant cannot exist for any length of time without fatty ingesta of some kind. Again, the incipient marasmic condition, or that already established, cannot be relieved unless a sufficient amount of assimilable fats, yielding but insignificant amounts of volatile fatty acids, is added to the nutriment. Vegetable oils and those animal fats exhibiting a high melting point being out of question, there is nothing left for us but to resort to the fats furnished by the yolk of the hen's egg. Yolk-fat, indeed, is the ideal fat for infants suffering from chronic gastro-intestinal disturbances, together with latent or even pronounced athrepsia infantum.

It would lead me too far to dwell again on the peculiar adaptability of the yolk of the hen's egg in the treatment of various forms of malassimilation. On this occasion I only wish to reiterate some of the factors which prompted me to substitute yolks for milk-fat in the treatment of undernourished infants afflicted with gastro-intestinal disease.

I. Yolk-fat, in its native state, in suitable amounts and admixture, is well-borne and well-liked by the majority of infants.

II. The great absorbability of yolk-fat; the residue left by yolk-fat in the feces, is smaller than that of any other animal fat.

III. The fat-components of the yolk of the hen's egg, palmitin, stearin and olein, yield no, or hardly any, volatile fatty acids, and consequently give no occasion to the production of the acetone bodies.

IV. The large amount of lecithin contained in the yolk tends to the restoration of nerve force, and acting as a general reconstituent ameliorates the cachectic condition.

V. The occurrence in the yolk of a diastatic ferment assisting in the conversion of amyloid substances.

VI. The property of the yolk to stimulate the digestive secretions.

The entire egg has been frequently employed by the pediatricist, the yolk alone but rarely. There is no consensus of opinion as regards the digestibility of the whole egg in the infantile alimentary tract. Some extol the egg as an important and readily digestible nutrient in early life, while others are absolutely opposed to its utilization.

Monti, who belongs to the latter class, deems it possible that the employment of storage-eggs to a certain extent may be responsible for his personal adverse opinion. He thinks that it is always risky to give eggs to children before they are weaned, as digestive disturbances are likely to be set up in the majority of instances.

There is no doubt that the total egg is badly borne by the average nursling. This finds its

analogue in a certain proportion of healthy adults who have an "idiosyncrasy" for eggs. The individual constitutional aversion to eggs in my opinion is solely due to the white of the egg. I have never met with it when raw, fresh yolks alone were partaken of. The white of the egg, which exhibits the bulk of the latter's protein substances, is apt to yield undue amounts of hydrogen sulphid and ammonia. This is particularly liable to occur in instances of retarded digestion of whatever causation. The yolk as previously shown leaves the stomach in a very short time and does not contain the elements giving rise to such amounts of hydrogen sulphid that it could produce any untoward results. A mixture of yolks with water and sugar, which has been recommended as a food for the newborn, is rightfully condemned by Monti. Such a combination is in the first instance irrational in itself, and there is no reason whatsoever why it should be given as a nutriment to the normal infant when it first enters life.

It must be understood once for all that yolks should neither find employment in the newborn nor in the infant which thrives on the physiological nutriment or on a modification of cow's milk. I suggest the use of yolks only in such pathologic conditions which may lead to *athrepsia infantum*, and which are due to or aggravated by the fat constituents of the nourishment.

There are two essentials which must be fol-

lowed when good shall result from the ingestion of yolks.

First.—The yolk-fat must completely replace the milk-fat.

Second.—The amount of yolk-fat, without being in excess, must be adequate, that is, it must conform to the caloric and nutritive demands of the organism.

The first essential is self-evident. By withholding the milk-fat from the nutriment we remove the eventual etiologic or aggravating factor of the underlying disease, or a fat-compound which, in the specific instance, has demonstrated its inadequacy in supplying the systemic demands. Substituting for it yolk-fat, we furnish to the deteriorated or diseased infantile organism a fat-combination which does not yield butyric acid or its derivatives, and which it is able to be anabolized in the great majority of instances. Cod-liver oil, empirically prescribed for undernourished children since time immemorial, although frequently serving a good purpose, will never be considered part and parcel of the infants' nutriment, and does not bear comparison with the yolk-fat as a dietetic factor. Untoward results obtained by yolks in the treatment of the undernourished, and more especially of athrepsia infantum, in a great measure are due to the non-withdrawal from the nourishment of the mischievous milk-fat.

The second essential—for all practical pur-

poses—is readily executed. A marasmic infant, in spite of abundant milk-fat, may continue to decline. It is evident, therefore, that the child does not properly anabolize the fatty substances introduced by the milk-fat. When this is replaced by another fat-compound, as yolk, for instance, it is not the amount of the latter but its physico-chemical constitution and its absorbability which primarily count. A very small amount of yolk, probably a quarter of a teaspoonful for each feeding, may be all that is needed when the change is first made. No matter how little of the proper fat-compound is ingested it serves a better purpose than a superabundance of the wrong fatty material. Even if during the first two or three weeks of yolk administration the child holds but its own, we have an indication that active decline has been checked for the time being. In everyday practice all that seems necessary when resorting to yolk-fat in the dietetic management of marasmic children is to start with aforementioned quantity and to very slowly increase it to the physiologic requirements of each individual case. The physiologic demands are satisfied when the digestive disturbance abates and the infantile organism thrives in a normal manner. These requirements, of course, are different in every instance and have to be ascertained in each individual case.

The fat-compound “yolk” differs widely from the fat-compound “cream.” Again, amount and

proportion of the fatty contents of the yolk are changeable as they are in the mammary secretion of the woman and the domesticated animals. Hence, the corresponding caloric and nutritive value of both fat-compounds cannot be accurately calculated.

The petroleum-ether extract of the yolk, according to Jean (*Annal. de Chimie Analyt.*, 8), amounts to 48.24 per cent.; the average quantity of fat in cream is about 20 per cent. An average yolk weighing about 12.5 grams, hence, contains about 6 grams of fat. The same amount of fat is exhibited in 31.25 c.c. of cream. Roughly speaking, an ounce of cream would, therefore, correspond to one yolk as regards their fatty contents, and a teaspoonful of yolk (4 grams) and 10 c.c. of cream would contain about equal amounts of fat.

A 1-per-cent. fat mixture is furnished approximately by 5 c.c. cream, or 2 grams yolk ($\frac{1}{2}$ teaspoon).

A 2-per-cent. fat mixture is furnished approximately by 10 c.c. of cream, or 4 grams of yolk (1 teaspoon).

A 3-per-cent. fat mixture is furnished approximately by 15 c.c. of cream, or 6 grams yolk ($1\frac{1}{2}$ teaspoons).

A 4-per-cent. fat mixture is furnished approximately by 20 c.c. of cream, or 8 grams yolk (2 teaspoons).

The primary caloric value of the fats of both

fat-compounds naturally is the same, but the absorbability of the yolk-fat as shown by me in the previous chapter ("The Yolk Cure in the Treatment of the Underfed") is greater than that of any other animal fat. As a matter of fact, therefore, yolk-fat generates more calories and is of higher nutritive value than milk-fat. Thus, it evinces that the figures heretofore given, can serve for general orientation only.

I do not offer any suggestions as to the further modification of cow's milk besides that on which I have already dwelt. There are numerous methods in vogue and every practitioner of experience knows how to obtain a certain percentage and quantity of proteids and carbohydrates. Practically speaking, I confine myself to the recommendation of skimmed milk* and the addition thereto of physiologic amounts of a non-deleterious native fat-compound. It is the office of the clinician to devise any further modification of the skimmed milk.†

The unaltered native yolk-fat is the one which I have made use of in my experiments. Good results can only be obtained by employing the un-

* Of course, skimmed milk still contains about 0.2 per cent. fat. This we cannot remove without rendering the milk unfit for the use of the infant.

† Whey and junket from modified skimmed milk and proportional amounts of egg yolk suitable for marasmic infants may be readily prepared. The milk-curdling ferment, however, should be in a menstruum free from alcohol and free acid, which latter are apt to increase the underlying gastro-intestinal disturbance.

manipulated raw yolk.* The less the yolk is agitated the easier it is of digestion, and the more it retains its fermentative and stimulating properties. It is to be added to the already prepared skim-milk modification after the latter has been warmed to about 105 deg. F.

The electrical conductivity of skimmed milk plus physiological amounts of yolk-fat is probably somewhat greater than that of native milk. Skimmed milk exhibits a greater conductivity than the native product, the former 48.57, the latter 47.62. This is caused by the interference of the fat globules with the movement of the ions. The fat-globules of native milk outnumber those which are due to yolk-fat when added in physiological amounts to skimmed milk.

Let us review the data which we may glean from this chapter.

First.—The overwhelming majority of cases of infantile marasmus occur in artificially nourished children.

Second.—The gastro-intestinal disturbances underlying infantile atrophy are very often due to the character of the food and not infrequently to its fatty contents.

Third.—While the *quantity* of fat aliment has found frequent practical consideration, the *chemical character* of the fatty substances entering into

* Cooking or sterilization changes the physical conditions of all animal fats, especially of that of the milk and yolk.

the baby's nutriment have hardly ever been inquired into by the clinician.

Fourth.—The composition of the fat of cow's milk is greatly at variance with that of the fat of human milk, differing especially in its far greater contents of volatile fatty acids among which butyric acid is the most important.

Fifth.—Butyric acid is the mother substance of the acetone bodies to the presence of which a number of disorders, to which the infant is prone, have been ascribed by various observers.

Sixth.—Butyric, caproic, caprylic and capric acids are contained in the fat of cow's milk in from six to eight times the amount in which they are present in that of human milk, and the large quantity of their respective ketones that is yielded in the alimentary tract must be a source of irritation and intoxication.

Seventh.—The infantile organism cannot cope successfully with the fat of cow's milk even in a mere physical sense. This is evidenced by the decidedly smaller absorption of the fat-compound derived from cow's milk than from human milk. The occurrence in the feces of absolutely and relatively larger amounts of fat of cow's milk is *prima facie* evidence of its more incomplete utilization by the youthful organism.

Eighth.—As the physical and chemical properties of the milk-fat are dependent upon the absolute and relative amount of lower and higher and uncombined fatty acids, it is evident that the

vast discrepancy existing between the constitution of cow's milk-fat and mother's milk-fat cannot be overcome by any possible modification of the former.

Ninth.—Apart from the butyric acid origin of the acetone bodies we find that the volatile fatty acids as furnished by the fat of cow's milk and their respective ketones are decided irritants of the delicate intestinal mucosa of the infant, and may also play the part of intoxicants. The ingestion of these acids is, therefore, the primary cause of many instances of gastro-intestinal irritation and disease followed by undernutrition, bodily retrogression and athrepsia infantum.

Tenth.—Alteration in the fat supply as exercised today is almost without exception a quantitative one, consisting of reduction, suspension and even increased supply of fat-aliment.

Eleventh.—Withdrawal of milk-fat in hand-fed infants frequently results in cessation of the local and systemic disturbance. It is, however, obvious that the infant cannot exist for any length of time without fatty ingesta of some kind. Furthermore, the incipient marasmic condition cannot be relieved unless a sufficient amount of assimilable fats yielding but insignificant amounts of volatile fatty acids is added to the nutriment.

Twelfth.—Yolk-fat seems to be the ideal fat for infants suffering from chronic gastro-intestinal disturbance together with latent or even pronounced athrepsia infantum.

Thirteenth.—Yolks should not find employment in the newborn nor in the infant which thrives on the physiologic nutriment or on a modification of cow's milk. Yolks should be used only in those pathologic conditions which may lead to athrepsia infantum and in those which are due to or aggravated by the fat constituents of the nourishment.

Fourteenth.—There are two essentials which must be followed in order to obtain good results from the ingestion of yolks, viz., the yolk-fat must completely replace the milk-fat, and the amount of yolk-fat, without being in excess, must be adequate, that is, it must conform to the caloric and nutritive demands of the organism.

Fifteenth.—The electrical conductivity of skimmed milk plus physiological amounts of yolk-fat is probably somewhat greater than that of native milk.

REFERENCES

¹¹ Archiv. f. Kinderheilkunde, Vol. II, 1881.

¹² Zeitschr. f. klin. Medizin., Vol. XLII.

ASSAY OF THE URINE

ASSAY OF THE URINE

The examination of the urine, as hereafter outlined, is limited to the substances which are found, or are apt to be found, in the renal secretion of the diabetic. Only the ordinary and most frequent pathological substances, and such simple tests which can be performed without especial technical skill and complicated, costly apparatus are mentioned. Qualitative and quantitative tests are given. In general, qualitative testing suffices in cases when the patient whose urine has been rendered free from pathologic bodies continues to remain under the same dietary influences; quantitative testing, on the other hand, becomes imperative when the pathologic material persists in the urine or when the slightest dietary change has taken place. Qualitative tests, of course, demand the least skill and time. Everybody, however, who devotes sufficient attention to it, may become well versed in the quantitative testing for the urinary substances that are of common occurrence in diabetes.

SUGAR

A. Detection

The delicacy of a test for glucose is dependent upon the character of the latter's solution, hence,

a test for dextrose, in point of sensitiveness, responds differently if applied to a watery solution than if used with the urinary fluid. Furthermore, the delicacy of a glucose test is not the same in all urines, it being dependent on quality and quantity of the other urinary constituents. Sensitiveness of a method for the detection of glucose in the urine is by no means identical with its accuracy and reliability. Thus, Trommer's test, for instance, is an exceedingly delicate one, if skilfully applied to certain urines; in the great majority of instances, however, its trustworthiness cannot be relied upon for amounts of dextrose less than 0.5 per cent. The same may be said of the other testing methods in general usage. Böttger's or Johnson's picric acid or the fermentation tests are equally delicate under favorable conditions, but, according to my experience, their employment should be restricted to urines containing 0.5 per cent. and more of grape-sugar.

By the application of the tests most generally used, pathologic quantities of urinary glucose may be overlooked altogether, or grape-sugar may be confounded with a number of other urinary constituents. Especially is this the case where the other concomitant manifestations are equally ill-pronounced. I have prepared the following table to show the limit of sensitiveness and that of reliability of the different tests.

Tests for Dextrose in the Urine.	Limit of Sensitiveness. Percentage.	Limit of Reliability. Percentage.
<i>Bismuth Tests.</i>		
Böttger's.....	0.1	0.5
Nylander's.....	0.1	0.3
Brücke's.....	0.2	0.4
Maschke's	0.2	0.4
<i>Copper Tests.</i>		
Trommer's.....	0.1	0.5
Salkowski's.....	0.5	0.5
Seegen's (filtration of urine through animal charcoal).....	0.01	0.05
Fehling's.....	0.3	0.3
Worm-Mueller's.....	0.25	0.3
Haines'	0.2	0.3
Elliott's.....	0.2	0.3
Pavy's.....	0.2	0.3
Phenylhydrazin test.....	0.03	0.05
Moore-Heller's.....	0.5	0.75
Penzoldt-Rubner's (ammonia-lead ace- tate).....	0.1	0.2
Methylene-blue test.....	0.04	0.3
Picric-acid test.....	0.1	0.5
Sodium sulpho-indigotin.....	0.1	0.5
Hoppe-Seyler's (Nitrophenylpropionic acid).....	0.5	0.75
Penzoldt's (Paradiazobenzolsulphonic acid).....	0.1	0.3
Furfurol tests.....	0.02	?
Horsley-Pratesi's (caustic potash, po- tassic silicate, potassic bichromate)	0.4	0.75
Fermentation test.....	0.1	0.5
Polarization.....	0.01	1.0
	with best instrument.	and over by instruments in general use.

NYLANDER'S TEST.—Almén's solution is the reagent employed in this method. It is prepared as follows: Potassium and sodium tartrate, 4 grams, and basic bismuth nitrate, 2 grams, are, under heat, dissolved in 100 grams of an 8 per cent. solution of sodium hydrate. When cooled the yellow bismuth oxid is filtered off and the solu-

tion, which does not deteriorate for a long time, is kept in a dark bottle. One part of the reagent is added to ten parts of the suspected urine. On boiling for from two to five minutes the white precipitate which is produced by the addition of the test solution to the urine, gradually assumes a more pronounced discoloration, turning finally black in the presence of dextrose. A very small percentage of sugar produces a brown hue of the precipitate; a dirty yellow or a grayish discoloration is of no significance as to the presence of dextrose. A secondary blackening of the precipitate, which may take place when the urine mixture in the test tube is cooled off, does not evince the occurrence of abnormal quantities of grape sugar. Uric acid and kreatinin do not reduce the bismuth salt. Pure peptone does also not influence the reaction. The test boils over easily; this may be prevented by placing the test tube into a water-bath.*

Disturbing factors and sources of error.—Pigmentary bodies, principally uroerythrin, hemato-porphyrin, indoxyl-glycuronic acid and other glycuronic acid compounds, ammonium carbonate in decomposing urines, hydrogen sulphid, mucin, albumin and other urinary constituents into whose composition sulphur enters. If the re-

* The reduction or pseudo-reduction of the bismuth salt may be frequently due to substances other than dextrose. *The chief value of this test, however, lies in its negative result, for the non-reduction of the bismuth positively excludes the presence of glucose in abnormal quantities.*

agent be added to a 0.6 per cent. albumin solution a red precipitate will be produced; in urine containing 1 to 2 per cent. of albumin the ensuing precipitate is black, and in appearance easily mistaken for the reduced bismuth salt. The reaction is still a fair one if 0.2 per cent. albumin occur together with 0.1 per cent. sugar; it is but faint if 0.35 per cent. albumin occurs simultaneously with the same quantity of sugar; if the reaction will not take place, 0.45 per cent. albumin is contained in a 0.1 per cent. sugar solution. Saccharin is among the substances preventing the reduction. Reduction of bismuth oxid has been noticed after the administration of a number of drugs, as antipyrin, acetanilid, arsenic, salicylic acid or salicylates, quinin, sulphur, mercurials, salts of iodine, turpentine, kairin, rheum, senna, santonin, tannic acid, chloral hydrate, tincture of eucalyptus, salol, benzol, sulfonal, trional, sodium benzoate, large doses of creosote (Stern), podophyllotoxin (Stern). There is little doubt, in my opinion, that the administration of some of these drugs is followed by an increased output of urinary pigmentary substances to which the reducing qualities have to be ascribed. In other instances, as with rheum and senna, the reducing power seems to lodge with the newly ingested chrysophanic acid ($C_{15}H_{10}O_4$).

Prevention.—Separation of the coloring matters, removal of the hydrogen sulphid by agitation with lead oxid, elimination of proteids.

Specimen of urine should be voided in a period during which no medicinal agents were administered.

For all practical purposes Nylander's method for the detection of sugar in the urine is the most simple and at the same time the most reliable test devised for the purpose. Nevertheless, the great value of this test is virtually but an indirect one—*the non-reduction of the bismuth salt is positive evidence of the absence of pathologic amounts of grape-sugar.*

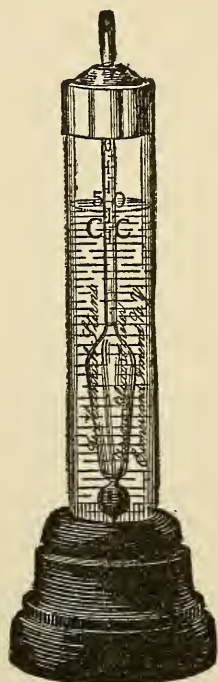
B. Determination

FERMENTATION TEST.—This is one of the most reliable tests for the detection of urinary glucose provided the latter occurs in quantities above 0.5 per cent. Certain vegetable organisms by their vitality call forth disintegration of grape-sugar. The products of decomposition vary according to the species of organism present. Beer-yeast breaks up the sugar molecule into carbon dioxid and alcohol in neutral or faintly acid solutions. In the main the fermentation occurs thus: $C_6H_{12}O_6 = 2C_2H_6O + 2CO_2$. Lactic acid yeast converts glucose into lactic acid; lactic fermentation may take place in diabetic urine if ammonium carbonate has replaced the latter's carbamid (urea). Butyric acid yeast converts sugar into butyric acid. Mycoderma aceti causes conversion of dextrose into an acid identical with glucinic acid. Kefyr produces

fermentative changes of glucose. A number of other specific microorganisms cause specific fermentations. The fermentation test for urinary glucose is made with fresh beer-yeast, or with dried German yeast, a combination of beer-yeast and dry starch, at a constant temperature of 33 to 35 C. For qualitative testing the fermentation tubes devised by Fritz Moritz* suffice. The determination of dextrose by collecting and measuring the carbon dioxid evolved during the process of fermentation may be approximately done with Einhorn's or Fiebig's saccharometers. *The method of determining the amount of glucose by the evolved carbon dioxid must always remain inexact, as the yeast per se, no matter what precautions are taken, furnishes small amounts of alcohol and carbonic acid gas. Einhorn's apparatus presents a further disadvantage in its open bulb, which permits the escape of considerable quantities of carbon dioxid.* The detection and determination of glucose by Robert's method—by ascertaining the density of urine before and after fermentation and by calculating the urine's contents of sugar by the loss of specific gravity during fermentation—is by far the most practical and trustworthy means in the hands of the general practitioner. The author has devised an apparatus for the practical and convenient application of this method. This apparatus, the glucosometer, may, moreover, be utilized for the

* Münchener Med. Wochenschrift, 1891, Nos. 1 and 2.

detection of glucose only, by interrupting the fermentation after an hour or so and by ascertaining any decline in the specific gravity of the dextrose solution.



STERN'S GLUCOSOMETER (One-half the Apparatus)

Disturbing factors and sources of error.—Maltose, fructose, and similar carbohydrates ferment with commercial yeast. The process, however, is a much slower one. It happens occasionally that a urine containing glucose is not fermentable. This is due to anti-fermentative agents, as salicylic acid, salol, arbutin, extractum myrtilli, etc., after their internal administration.

Directions for Using Stern's Glucosometer.—

The urine is placed into the fermentation tube exactly to the mark 50 c.c. Then the desired urinometer (two such instruments accompany the apparatus, one for the specific gravity from 990 to 1030, the other from 1030 to 1070) is inserted and the aluminum cap is placed in its proper position. Following this the specific gravity on the flat top of the cap is read off and written down. (The reading of the specific gravity on the flat top outside the tube fixes the meniscus and permits exactness; half a degree of the specific gravity can be readily estimated.) After this cap and urinometer are removed and three or four grams, that is one-quarter or one-third of a cake of Fleischmann's yeast is added. Again the same hydrometer is inserted and the tube closed by means of the cap. (The hydrometer need not be placed into the tube at once; this can be done after fermentation is completed. However, it is better to place it in position when starting, as it will then indicate, step by step, the progress of the fermentation.) The yeast cell will become active at a temperature of from 27 to 28 deg. C., that is from 80 to 82 deg. F. The apparatus should, therefore, be placed in a room in which there is a constant temperature of that degree. Fermentation in this moderate temperature will be completed in from twelve to fifteen hours. The CO_2 which is evolved during the process will escape through the perforations in the aluminum cap. After completion of the fermenta-

tion the cap should be lifted for a short time, the urine stirred or whipped, or the tube shaken to effect a more complete elimination of the CO_2 . (Care must be taken that not a drop of the urine be spilled.) After this the cap should be again placed in position, the yeast particles be allowed to settle (which takes place rapidly), and the specific gravity be read off at the flat top of the cap. The difference between the specific gravity before and after fermentation is then compared and the degrees of density lost during this process ascertained. Each degree of specific gravity lost corresponds to 0.2288 (approximately 0.23) gram of glucose in every 100 c.c. of urine (one grain to the ounce of urine). Thus, if the density of the unfermented urine was 1036 and of the fermented urine 1020, the sixteen lost degrees must be multiplied by 0.2288. Therefor, 3.6608 grams of glucose are contained in 100 c.c. of the urine.

The amount of glucose voided during the twenty-four hours may be easily calculated if the quantity of urine passed in that time be known and if the examined portion of the same was a specimen of the diurnal mixture. If it be desired to ascertain the quantity of glucose in grains to the fluid ounce of urine, no calculation is necessary, as each degree of density corresponds exactly to one grain of glucose per fluid ounce. For instance, if the specific gravity of the urine be 1036 before fermentation and 1020

after this process, sixteen grains of glucose were contained in each ounce of the tested urine.

To effect complete fermentation in six hours the tubes may be had in a form that they can be inserted into a sand-bath or used in the incubator. The best protectors and, incidentally, the best promoters of the yeast cell's activity, are the sand-bath or the incubator with a temperature at or near 33.5 deg. C. (92 deg. F.).

FEHLING'S TEST.—In case the urine contains appreciable amounts of serum albumin and globulin they should be removed by acidifying, boiling, and filtering before it is subjected to Fehling's test. However, the serines only give rise to a partial reduction of the test fluid, and it is hardly necessary to remove them in the ordinary examination.

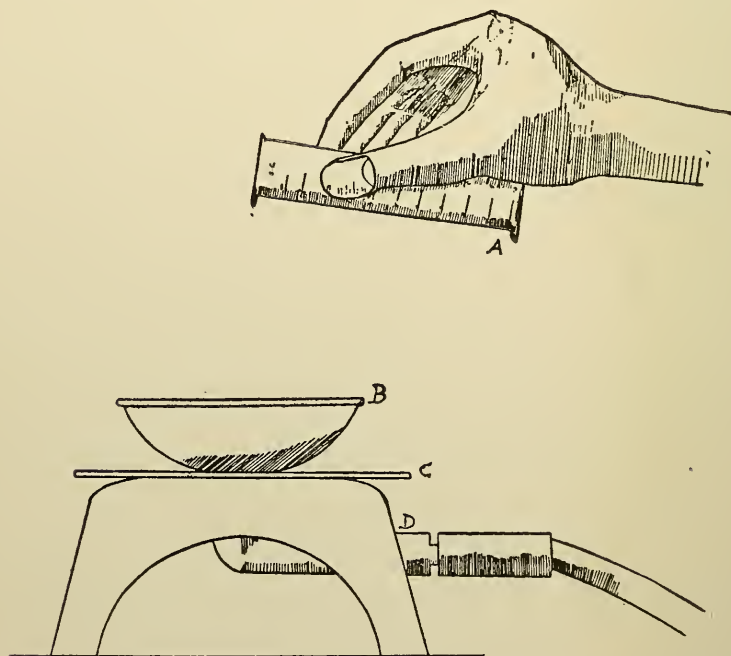
It is generally recommended that the diabetic urine be diluted ten times its volume with water before proceeding with the test proper. This, however, is only called for in cases when the sugar content is very large. For clinical purposes the test with the native urine will ordinarily give quite accurate results.

Preparation of Fehling's solution.—Solution A.: 34.64 grams sulphate of copper is dissolved in 500 c.c. of distilled water.

Solution B: 180 grams neutral potassium tartrate and 70 grams sodium hydrate are dissolved in 300 c.c. of water. When cold, 200 c.c. of water are added.

These solutions should be kept in separate bottles and mixed as required in equal volumes of 5 c.c. each. The copper in 10 c.c. of Fehling's solution is reduced by exactly 0.05 gram glucose.

Method.—Exactly 5 c.c. of each solution (A and B) is measured off in a 10 c.c. cylinder graduate. The 10 c.c. are placed into a porcelain evaporating dish which stands in a water-bath or on an asbestos or iron screen. Gas or alcohol is used for boiling.



APPARATUS FOR STERN'S MODIFICATION OF FEHLING'S TEST

- A. Cylinder graduate
- B. Evaporating dish
- C. Asbestos or iron screen (foil)
- D. Gas pipe

Following this the same narrow cylinder graduate (which is divided into tenths of cubic centimeters) is thoroughly cleaned and the urine filled exactly to the 10 c.c. mark. The urine is then allowed to drop slowly into the boiling Fehling's solution, while the mixture is being constantly stirred. This is continued until the blue or purplish color has entirely disappeared and until the mixture is uniformly yellowish or reddish and a slight deposit of either yellow or red has formed.

(The end reaction is the most difficult factor connected with the test. The assay should be conducted in daylight. One soon becomes an expert in testing with Fehling's solution if proper care is exercised in adding no more nor less urine to the test-fluid than is necessary to produce a uniform reduction. If the porcelain dish is left standing after the copper is reduced, the mixture soon turns blue again. This is due to the hydration of the copper and has nothing to do with an insufficient reduction.)

In case the solution is not reduced by 10 c.c. of the urine, an additional 10 c.c. urine are poured into the cylinder graduate, and this is repeated until 30 or 40 c.c. urine are added to the boiling Fehling's solution in the porcelain dish (a dish of 70 c.c. capacity is the best for ordinary use).

Now the amount of urine that was necessary to effect the complete reduction of the solution

is ascertained, and the amount of urinary sugar calculated.

Example.—If 6 c.c. of undiluted urine have been required to reduce all the copper in the 10 c.c. Fehling's solution, then the 6 c.c. urine contain 0.05 gram glucose.

Expressed in per cent., this is

$$6:100::0.05:X = 0.83$$

The twenty-four hours' output of sugar is readily calculated by multiplying the per cent. figure by the number of cubic centimeters of urine voided in this period. If, for instance, 2150 c.c. of urine were passed by the same patient during this time, then $0.83 \times 2150 = 17.84$ grams glucose were excreted by him.

The accompanying tables will be found very useful in the clinical laboratory:

TABLE SHOWING THE EQUIVALENCE OF GRAMS PER CENT. OF SUGAR IN GRAINS PER OUNCE OF URINE.

Grams per cent.		Grains per ounce.		Grams per cent.		Grains per ounce.
0.05	=	0.218		0.8	=	3.498
0.1	=	0.437		0.85	=	3.717
0.15	=	0.656		0.9	=	3.937
0.2	=	0.8746		0.95	=	4.156
0.25	=	1.093		1.0	=	4.375
0.3	=	1.312		1.1	=	4.8
0.35	=	1.530		1.2	=	5.25
0.4	=	1.749		1.3	=	5.70
0.45	=	1.968		1.4	=	6.125
0.5	=	2.186		1.5	=	6.561
0.55	=	2.406		1.6	=	7.00
0.6	=	2.624		1.7	=	7.437
0.65	=	2.843		1.8	=	7.874
0.7	=	3.062		1.9	=	8.311
0.75	=	3.280		2	=	8.750

Grams per cent.		Grains per ounce.		Grams per cent.		Grains per ounce.
2.5	=	10.936		6.5	=	28.436
3	=	13.125		7	=	30.622
3.5	=	15.311		7.5	=	32.808
4	=	17.500		8	=	34.994
4.5	=	19.686		8.5	=	37.183
5	=	21.872		9	=	39.370
5.5	=	24.058		9.5	=	41.558
6	=	26.250		10	=	43.750
6.25	=	27.343				

TABLE SHOWING PER CENT. OF SUGAR PRESENT IN THE URINE, WHEN THE FOLLOWING AMOUNTS OF PURE URINE HAVE BEEN REQUIRED TO COMPLETELY REDUCE THE COPPER IN 10 c.c. OF STANDARD FEHLING'S SOLUTION.

Amount of Urine used.		Grams of Sugar per cent.		Amount of Urine used.		Grams of Sugar per cent.
0.1 c.c.	=	50		2.25 c.c.	=	2.22
0.2 "	=	25		2.3 "	=	2.17
0.25 "	=	20		2.4 "	=	2.08
0.3 "	=	16.6		2.5 "	=	2
0.4 "	=	12.5		2.6 "	=	1.92
0.5 "	=	10		2.7 "	=	1.85
0.6 "	=	8.33		2.75 "	=	1.81
0.7 "	=	7.14		2.8 "	=	1.78
0.75 "	=	6.6		2.9 "	=	1.72
0.8 "	=	6.25		3 "	=	1.66
0.9 "	=	5.55		3.25 "	=	1.53
1 "	=	5		3.5 "	=	1.42
1.1 "	=	4.54		3.6 "	=	1.38
1.2 "	=	4.17		3.7 "	=	1.35
1.25 "	=	4		3.75 "	=	1.33
1.3 "	=	3.8		3.9 "	=	1.28
1.4 "	=	3.57		4 "	=	1.25
1.5 "	=	3.33		4.25 "	=	1.17
1.6 "	=	3.12		4.5 "	=	1.11
1.7 "	=	2.94		4.6 "	=	1.08
1.75 "	=	2.85		4.7 "	=	1.06
1.8 "	=	2.77		4.75 "	=	1.05
1.9 "	=	2.63		4.9 "	=	1.02
2 "	=	2.5		5 "	=	1
2.1 "	=	2.39		5.25 "	=	0.95
2.2 "	=	2.27		5.5 "	=	0.9

Amount of Urine used.		Grams of Sugar per cent.		Amount of Urine used.		Grams of Sugar per cent.
5.75 c.c.	=	0.87		10.5 c.c.	=	0.47
6 "	=	0.83		11 "	=	0.45
6.25 "	=	0.8		11.5 "	=	0.43
6.5 "	=	0.76		12 "	=	0.416
6.75 "	=	0.73		12.5 "	=	0.4
7 "	=	0.71		13 "	=	0.38
7.25 "	=	0.68		14 "	=	0.36
7.5 "	=	0.66		15 "	=	0.33
7.75 "	=	0.64		16 "	=	0.31
8 "	=	0.625		17 "	=	0.29
8.25 "	=	0.606		18 "	=	0.27
8.5 "	=	0.58		19 "	=	0.26
8.75 "	=	0.57		20 "	=	0.25
9 "	=	0.55		25 "	=	0.2
9.25 "	=	0.54		30 "	=	0.16
9.5 "	=	0.52		35 "	=	0.14
9.75 "	=	0.51		40 "	=	0.125
10 "	=	0.5		50 "	=	0.10

ACETONE

Detection

(a) SODIUM NITROPRUSSIDE TEST.—Reagent: Solution of 0.1 gram sodium nitroprusside in 15 c.c. distilled water.

Method.—To 2 c.c. of the solution contained in a test tube twice the volume of freshly voided urine is added. After the addition of some potassium or sodium hydrate a ruby red color is assumed by the mixture in case acetone be present. On the addition of a few drops of acetic acid this color disappears at once, if acetone is not contained in the urine. If, however, acetone be present, the addition of acetic acid intensifies the ruby tint.

Sources of error.—Kreatinin, aldehydes and

chloroform may give similar reactions, but the red color quickly turns to yellow on the addition of acetic acid.

(b) SODIUM NITROPRUSSIDE TEST (NIECE'S IMPROVEMENT).—Reagent: Ammonium nitrate (crystals), 30 grams; sodium nitroprusside (crystals), 2 grams; distilled water, enough to make 80 c.c. Mix in the order given, dissolve and keep in a well-corked bottle protected from sunlight.

Method.—With 5 c.c. of freshly voided urine contained in a test tube 0.5 to 1 c.c. of the reagent is well mixed. Then 3 c.c. of a 10-per-cent. solution of ammonium hydroxid are carefully added by contact. If acetone be present there is immediately produced at the zone of contact a purple or "Burgundy red" coloration. On standing the color diffuses mostly upward into the ammonium layer; its depth of color is in proportion to the amount of acetone present.

When the amount of acetone be small the color is in the beginning a deep rose pink, but it soon changes to purple or violet. The mixture should be allowed to stand for from two to five minutes before examination. When the two solutions blend there ensues coloration of the entire fluid. The color deepens gradually. After another five or ten minutes the coloration has reached its greatest degree of intensity. The color slowly fades away after fifteen or twenty minutes, so that within one hour it has most always entirely disappeared.

The reagent keeps indefinitely, and the usual sources of error when employing the ordinary sodium nitroprusside test do not obtain. The action of the ammonium salt in the test is that of a stabilizer and catalyzer; that is, it prevents decomposition of the nitroprusside while in solution with water, assists in avoiding false side-reactions, and enhances the sensitiveness of the reagent.

(c) **LIEBEN'S TEST.**—Reagents: Solution of iodine in potassium iodide and solution of potassium hydrate.

Method.—To the urine (a distillate, if possible) in the test tube some potassium hydrate solution is added. To this is added some drops of a strong aqueous solution of iodine in potassium iodide. If acetone be present, crystals of iodoform will separate which appear under the microscope as six-sided plates or stars. This reaction is neither as sensitive nor as reliable as Niece's modification of the sodium nitroprusside test. The scarcity at this time (during the European war) of this chemical, however, compels one to resort to inferior and more complicated methods.

DIACETIC ACID

(Aceto-acetic acid)

Detection

FERRIC CHLORIDE TEST (GERHARDT).—To 5 c.c. of recently voided urine contained in a test tube

a strong solution of ferric chlorid is added drop by drop. The precipitated phosphates are filtrated off and to the filtrate a few drops more of the ferric chlorid solution are added. In case a mahogany-red color is produced, it is probably due to the occurrence of diacetic acid. This color disappears when the mixture is boiled, and it will not appear if the urine has been boiled prior to the application of the test.

AMMONIA

Determination

FORMALIN TEST.—Reagents: Formalin (40 per cent. commercial). Alcoholic solution phenolphthalein (1 per cent.) N/10 solution sodium hydrate.

Method.—Ten c.c. urine are diluted with 50 c.c. water. To this are added 2 or 3 drops phenolphthalein (1 per cent. alcoholic solution). After this the mixture is neutralized with N/10 sodium hydrate. Following this 5 c.c. formalin (40 per cent. commercial), previously neutralized with N/10 sodium hydrate, are added to the mixture, which is again titrated with N/10 sodium hydrate until a permanent faint pinkish color is produced.

Each cubic centimeter of the decinormal sodium hydrate used in the final titration is equivalent to 1 c.c. of decinormal ammonia, that is, 0.001704 gram of ammonia. By multiplying the number of cubic centimeters of decinormal so-

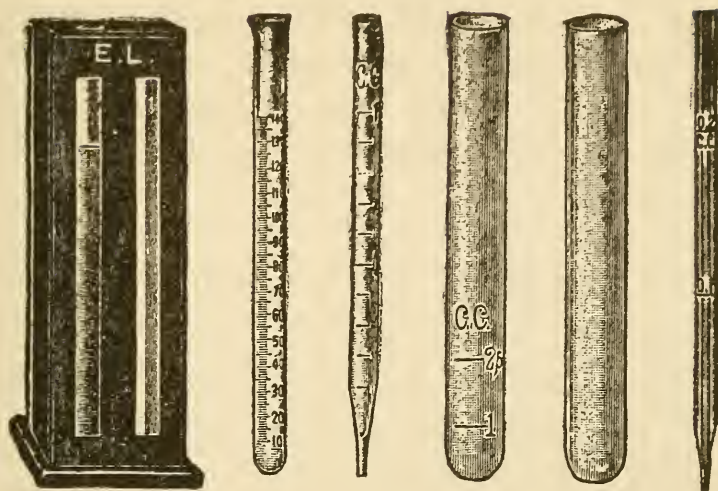
dium hydrate solution employed in the final titration with 0.001704, the amount of ammonia contained in the 10 c.c. of urine is obtained.

The method is not entirely free from sources of error, but is sufficiently reliable for clinical purposes.

ESTIMATION OF SUGAR IN THE BLOOD

EPSTEIN'S MICROCHEMICAL TEST.—Blood-sugar determinations are, as yet, comparatively rarely made in clinical work. The reasons of this are manifest. The methods commonly employed are cumbersome and time-consuming; the amount of blood usually withdrawn precludes the frequent application of the older tests, which entail an outlay on apparatus and reagents that cannot always be defrayed by the private physician. Epstein has adopted the Sahli-Gowers hemoglobin colorimeter (with suitable color standards) for the determination of sugar in the blood. This colorimeter is not an expensive instrument, and it enables the examiner to work with very small amounts of the necessary reagents and blood. As a matter of fact, one can with its aid estimate with precision the quantity of sugar present in 0.2 c.c. or even 0.1 c.c. of blood.

Reagents: (a) Picric acid, saturated solution; (b) sodium carbonate, 10 per cent. solution; (c) sodium fluorid or potassium oxalate, 2 per cent. solution.



EPSTEIN'S OUTFIT FOR ESTIMATING SUGAR IN THE BLOOD BY
MICROCHEMICAL METHOD.

1. Sahli-Gowers hemoglobinometer stand and graduated tube. 2. Two standard color tubes, one (*A*) suitable for measuring quantities of sugar in the blood ranging from 0.05 per cent. to 0.1 per cent.; the other (*B*) is adapted to quantities of sugar over 0.1 per cent.; 3. A special pipet (resembling but larger than the hemoglobin blood-pipet) graduated at 0.1 c.c. and 0.2 c.c. for collecting and measuring the blood. 4. A test tube ($\frac{1}{2}$ by 4 inches) graduated at 1.0 c.c. and 2.5 c.c. 5. Another test tube of similar dimensions (not graduated) suitable for boiling.

(The tubes belonging to this hemoglobinometer are not all equally calibrated. With some the 50 per cent. mark represents a volume of 1.0 c.c.; with others, 1.0 c.c. of fluid reaches up to the 43, 45, 46 or 47 per cent. mark. In all of the tubes the error in the calibration is below the 10 per cent. mark; the graduations above this mark are usually correct. By means of the standard 1.0 c.c. pipet one can readily determine whether or not a given tube is properly calibrated. In order to facilitate a direct reading of the percentage of sugar on these hemoglobinometer tubes, it is essential to have 1.0 c.c. of fluid stand at mark 50. To overcome a discrepancy (if any exists) in the calibration of a given tube, one may put one, two or three small glass beads in the bottom of the tube, of such size as to raise the meniscus of 1.0 c.c. of fluid up to the 50 per cent. mark.

Method.—One or two drops of the sodium fluorid or potassium oxalate solution is placed into the graduated test tube (4). By means of the blood pipet (3), 0.2 c.c. of blood is obtained from the tip of the finger or the lobe of the ear and is released into the tube (4) in which the fluorid or oxalate solution is contained. The pipet is rinsed two or three times with distilled water and the washings added to the blood in the tube (4). After this distilled water is permitted to flow into the same tube to the 1.0 mark. When laking of the blood has ensued, picric acid, a few drops at a time, is added to it until the mark 2.5 c.c. is reached. As the picric acid is added the tube is gently shaken. Precipitation of the blood-proteins takes place. The sugar, together with an excess of picric acid sufficient for the reaction, stays in solution. The tube is finally shaken vigorously while its mouth is being covered with the finger. Following this the contents are filtered through a small filter, or, better still, centrifuged for one or two minutes.

One cubic centimeter of the filtrate or the clear supernatant fluid obtained on centrifuging is withdrawn, placed into the boiling tube (5) and heated carefully over the naked flame. The entire contents of the tube are boiled until but 2 or 3 drops of the solution are evaporated. One-half cubic centimeter of the 10 per cent. sodium carbonate solution is then added and the tube heated again until the contents are concentrated

to a small volume equal to about 2 or 3 drops. The color of the fluid changes from yellow to deep-red or reddish-brown, and the reaction is completed.

Three or four drops of distilled water are added and the tube warmed gently. The contents are then transferred to the graduated tube (1). The boiling tube is rinsed several times with water (using only 3 or 4 drops at a time). The tube is warmed with each rinsing before transferring the contents to the graduated tube. The volume is then made up to the mark 50.

The color of the resulting solution is compared with that of the two standard tubes, A and B. If it is darker than standard A (representing 0.05 per cent. sugar) and lighter than standard B (representing 0.1 per cent. sugar), the first standard is used for comparison. In either case, the solution in the graduated tube is diluted gradually with water (as in hemoglobin estimations) until the colors match as closely as possible.

The percentage of sugar in the blood is then computed thus: Using the lighter standard A, the figure on the scale, divided by 1,000, represents the percentage of sugar in the blood. For example, the tube reads 81, then the result is

$$\frac{81}{1,000} = 0.081 \text{ per cent.}$$

When standard B is used for comparison, the

figure on the scale is multiplied by 2 and divided by 1,000. For example, the tube reads 69, then the percentage of sugar is

$$\frac{69 \times 2}{1,000} = 0.138 \text{ per cent.}$$

With the instructions given, the above formulas may be used for direct computation of the percentage of sugar only, when 0.2 c.c. of blood is used in the determination. When, however, only 0.1 c.c. of blood is used, the formulas apply as well, but the value obtained must be multiplied by 2.

In cases in which a high sugar content in the blood is suspected, as in diabetes, it is best to use only 0.1 c.c. of blood for the determination. In all other cases 0.2 c.c. of blood should be employed.

NUTRITIVE CONSTITUENTS OF FOOD

The following tables, taken almost in their entirety from the well-known works of J. König, are given for general orientation only. The same foodstuffs, grown under different conditions and surroundings, may greatly differ from each other in their percentage composition. Their assay—at the best—can furnish definite information concerning only those specimens subjected to examination. Again, in the milder forms of diabetes, too much consideration should not be attached to this factor. In the graver types of the disease, however, this must be taken into due account.

Nitrogenous matter comprises the proteins, albumins, animal and vegetable caseins, gelatinous and glutinous substances.

Nitrogenous-free extractive matter (carbohydrates) includes starch, sugar, gums, dextrin, pectin, alcohol and cellulose.

Foodstuffs, whose names appear in italics, are rich in sugars and starches. They must not be used by the diabetic without permission of his physician.

	Nitrogenous Matter	Fat	N-Free Extractive Matter (Carbohy- drates)
<i>Meat and Fish</i>			
Beef, fat.....	17,19	29,28	—
“ medium fat.....	20,91	5,19	0,48
“ lean.....	20,78	1,50	—
Cow's meat, fat.....	19,86	7,70	0,41
“ lean.....	20,64	1,78	0,01
Veal, fat.....	18,88	7,41	0,07
“ lean.....	19,86	0,82	—
Mutton, fat.....	14,80	36,39	0,05
“ half fat.....	17,11	5,77	—
Pork, fat.....	14,54	37,34	—
“ lean.....	20,25	6,81	—
Blood.....	18,12	0,18	0,03
Lung.....	14,74	4,57	1,00
Heart.....	17,71	8,66	0,64
Kidney.....	18,01	5,11	0,15
Spleen.....	17,77	4,19	1,50
Tongue.....	14,29	17,18	0,51
Liver, ox.....	19,72	5,55	1,69
“ calf.....	17,66	2,39	5,47
Sweetbreads (thymus).....	28,00	0,40	—
Ham, ordinary.....			
“ lean (smoked).....	25,08	8,11	—
Westphalian (smoked).....	24,47	36,45	0,16
Smoked beef.....	27,10	15,35	—
“ ox tongue.....	24,31	31,61	—
“ goose breast.....	21,45	31,49	1,15
Tinned beef.....	19,41	13,07	—
“ tongue.....	15,35	15,14	2,01
American canned beef.....	29,04	11,54	—
Goose liver paste.....	14,59	33,59	2,67
Beef paste.....	17,17	44,63	3,36
Ham paste.....	16,88	50,88	—
Tongue paste.....	18,46	32,85	0,46
Salmon paste.....	18,48	36,51	0,70
Lobster paste.....	14,87	24,86	4,05
Anchovies paste.....	12,33	1,59	5,18
Cervelat sausage.....	17,64	39,76	—
Blood sausage.....	11,81	11,46	25,09
Liver “.....	15,93	26,33	6,38
Headcheese.....	23,10	22,80	—
Knackwurst.....	22,80	11,40	—
Frankfurt sausage.....	11,69	39,61	2,25
Meat sausage.....	27,31	39,88	5,10
Ham sausage.....	12,87	24,43	12,52

	Nitrogenous Matter	Fat	N-Free Extractive Matter (Carbohy- drates)
<i>Meat and Fish (Continued)</i>			
Truffle sausage.....	13,06	41,27	—
Bacon, smoked, American.....	9,27	75,75	—
“ “ German.....	2,60	77,60	—
Rind of pork.....	35,32*	3,75	—
Calves' feet.....	23,00*	11,32	0,70
Bones.....	15,50*	0,50	—
Hare.....	23,34	1,13	0,19
Venison.....	19,77	1,92	1,42
Rabbit.....	21,47	9,76	0,75
Horse.....	21,71	2,55	0,46
Chicken, fat.....	18,49	9,34	1,20
Rooster, young, lean.....	23,32	3,15	2,49
Duck, wild.....	22,65	3,11	2,33
Squab.....	22,14	1,00	0,76
Partridge.....	35,26	1,43	—
Fieldfare.....	22,19	1,77	1,39
Turkey, medium fat.....	24,70	8,50	—
<i>Fats</i>			
Butter, ordinary.....	1,09	77,48	0,47†
“ fine.....	0,28	85,50	0,69†
Ox fat.....	0,44	98,15	—
Lard.....	0,26	99,04	—
Fat tissue.....	2,27	85,43	—
<i>Fish</i>			
Sole.....	11,94	0,25	0,45
Codfish.....	16,23	0,33	—
Haddock.....	17,09	0,34	—
Perch.....	18,11	0,44	0,01
Pike.....	18,34	0,51	0,63
Flounder.....	14,03	0,69	—
Ray.....	19,51	0,91	—
Carp.....	21,86	1,09	—
Plaice.....	19,98	1,80	—
Sturgeon.....	18,00	1,90	—
Trout.....	19,18	2,10	—
Gudgeon.....	17,37	2,68	—
Smelt.....	19,36	4,92	—
Sea eel.....	13,57	5,02	0,39
Halibut.....	18,53	5,16	—
Salmon.....*	15,01	6,42	2,85

*Chiefly gelatinous matter.

†Sugar of milk.

Vegetable oils are pure fats.

	Nitrogenous Matter	Fat	N-Free Extractive Matter (Carbohydrates)
<i>Fish (Continued)</i>			
Herring	10,11	7,11	—
Mackerel	19,36	8,08	—
Blay	16,81	8,13	—
Shad	18,76	9,45	—
River eel	12,83	28,37	0,53
Salt herring	18,90	16,89	1,57
Sardelles, salted	22,30	2,21	—
Codfish, salted	27,07	0,36	—
Mackerel, salted	19,17	22,43	0,13
Bloater, smoked	21,12	8,51	—
Sprats	22,73	15,94	0,98
Lamprey	20,18	25,59	1,61
Sardines, canned	25,90	11,27	0,19
Caviar	30,79	15,66	1,67
Haddock (dried)	} 76,07	0,70	—
Codfish (dried)			
Oyster	9,04	2,04	6,44
Clam	8,69	1,12	4,12
Lobster, fresh	14,49	1,84	0,12
“ canned	18,13	1,07	0,58
Crayfish, fresh	13,63	0,36	0,21
“ canned	16,10	0,46	1,01
Crab, fresh	15,80	1,54	0,75
“ canned	25,38	1,00	0,24
<i>Milk and Dairy Products</i>			Sugar* of milk
Cow's milk	3,41	3,65	4,81
Goat's milk	3,52	3,94	4,39
Sheep's milk	6,31	6,83	4,73
Butter	4,06	83,27	3,73
Whey	0,85	0,23	4,70
Sour milk	3,41	3,65	3,50
Cream	3,61	26,75	3,52
Kefir	3,26	0,86	2,04
Pot cheese	25,04	5,04	2,57
Lean cheese	34,99	11,37	5,40
Half fat cheese	27,24	23,71	1,54
Fat cheese	25,09	29,05	2,22
Cream “	16,28	41,22	1,90
Dutch “	29,48	26,71	3,72
Edam “	24,07	30,26	4,48
Limburger	25,09	29,05	3,70
Emmenthal	32,42	29,67	0,31

	Nitrogenous Matter	Fat	N-Free Extractive Matter (Carbohy- drates)
<i>Milk and Dairy Products (Cont'd)</i>			Sugar of milk
Mayence hand cheese.....	36,33	5,55	1,00
Hohenburg hand cheese.....	26,90	29,13	?
Parmesan cheese.....	41,19	19,52	1,18
Swiss cheese.....	23,90	22,54	5,04
Westphalian cheese (half fat)....	27,49	26,06	1,15
“ “ (fresh).....	29,85	11,76	7,98
Neufchâtel cheese.....	17,44	40,80	5,21
Cheshire cheese.....	27,68	27,46	5,89
Caraway seed cheese.....	31,61	7,36	10,43
<i>Eggs</i>			
Hen's egg, white.....	12,87	0,25	0,77
“ “ yolk.....	16,12	31,39	0,48
“ “ whole egg.....	12,55	12,11	0,55
“ “ “ “.....	14,49	15,82	0,62
1 egg 50 grams.....	6,30 g.	6,05 g.	0,27 g.
1 “ “ “.....	7,24 g.	7,91 g.	0,31 g.
Duck egg.....	12,24	15,49	—

	Nitrogenous Matter	Alkaloids Coffein, Thein	Fat	Sugar	N-Free Extractive Matter	Cellulose
<i>Alkaloidal Stimulants</i>						
Coffee, roasted.....	13.98	1.24	14.48	0.66	45.09	19.89
Tea.....	24.50	3.58	7.07	15.65* Starch	26.04	11.58
Cocoa.....	13.97	1.56	53.03	15.17	21.81	3.40
“ powdered, German.....	21.50	1.82	27.34	12.61	19.01	5.44
“ Dutch.....	19.66	1.74	31.61		17.25	5.85

*Tannic acid

	Nitrogenous Matter	Fat	Sugar	Dextrin	Starch, etc.	Cellulose
<i>Meals and Flour Preparations</i>						
Wheaten gluten flour.....	80.00	1.20		8.30		0.50
Wheat-bran.....	13.50	2.50		30.80		31.60
Wheaten flour, fine.....	10.21	0.94	2.35	3.03	69.30	0.29
“ coarse.....	12.06	1.36	1.86	4.09	65.88	0.98
Whole wheaten flour (Graham).....	11.70	1.70		69.90		1.90
Wheaten grist.....	9.42	0.94		75.92		0.21

	Nitrogenous Matter	Fat	Sugar	Dextrin	Starch, etc.	Cellulose
<i>Meals and Flour Preparations (Continued)</i>						
<i>Rye flour, fine</i>	10.21	1.64	3.89	73.54	58.66	0.64
“ <i>coarse</i>	11.57	2.08		7.16		1.59
<i>Barley grist</i>	7.25	1.15		76.19		1.36
“ <i>flour</i>	11.38	1.53	3.11	6.52	61.60	0.45
<i>Oatmeal</i>	13.44	5.92	2.26	3.08	61.67	1.86
<i>Corn meal</i>	9.65	3.80	3.56	3.36	62.63	1.46
<i>Rice flour</i>	6.91	0.67		78.84		0.18
<i>Millet flour</i>	7.61	3.41		72.56		1.24
<i>Buckwheat flour</i>	8.87	1.56	1.06	2.95	70.30	0.67
<i>Potato flour</i>	1.03	—		80.83		—
<i>Bean flour</i>	23.19	2.13		59.37		1.67
<i>Pea flour</i>	25.20	2.01		57.17		1.32
<i>Lentil flour</i>	25.46	1.83		57.35		2.01
<i>Soya bean flour</i>	25.69	18.83		38.12		2.75
<i>Starch (wheat)</i>	1.18	0.06		82.13		0.13
“ <i>(corn)</i>	1.53	—		84.00 incl. cellulose		—
“ <i>(potato)</i>	0.51	—		82.04		—
<i>Maizena</i>	0.47	—		84.94 incl. cellulose		—
<i>Sage</i>	0.81	—		86.11	“	—
<i>Sage flour</i>	3.75	—		77.88	“	—
<i>Tapioca</i>	0.63	—		85.95	“	—
<i>Arrowroot</i>	0.88	—		82.41	“	—
<i>Oat, groats</i>	11.29	6.67		70.30		0.91

	Nitrogenous Matter	Fat	Sugar	Dextrin	Starch, etc.	Cellulose
<i>Bread</i>						
Gluten bread.....	18.86	0.15	{	31.19	{	0.44
Gluten biscuits.....	76.37	2.00		10.53		—
Wheaten bread, fine, fresh.....	7.06	0.46	{	4.02	52.56	0.32
“ “ coarse.....	6.15	0.44		2.08	49.04	0.61
Roll.....	8.65	0.45		3.77	59.63	0.38
Water roll, crumb.....	6.50	1.00		2.48	40.32	8.89
“ “ crust.....	9.25	0.61		3.61	59.24	14.00
“ “ whole.....	6.15	0.91		1.86	40.33	6.95
Biscuit, German.....	11.93	7.47		36.38	32.29	0.75
“ English.....	7.18	9.28		17.02	58.08	0.16
Wheaten bread Zwieback.....	8.55	0.98		1.82	73.28	0.59
“ “ “ fine.....	13.31	3.18		7.12	76.96	0.25
Graham bread, fresh.....	9.00	1.00	{	50.00	{	0.32
Rye bread.....	6.11	0.43		2.31		46.95
Black bread, much crust.....	10.79	—	—	57.82	—	—
“ “ crumb only.....	8.30	—	—	44.20	—	—
Ammunition bread.....	7.47	0.45	3.05	46.36	1.51	1.51
Pumpernickel.....	7.59	1.51	3.25	41.87	0.94	0.94
Rye Zwieback.....	9.31	0.96	3.65	67.66	4.73	4.73
Oat Zwieback.....	8.39	6.03	4.09	60.12	5.82	5.82
Oat cakes.....	8.38	12.98	18.29	54.67	0.72	0.72
Barley Zwieback.....	9.33	1.09	4.66	64.40	4.29	4.29

	Nitrogenous Matter	Fat	Sugar	N-Free Extractive Matter	Cellulose
<i>Vegetables, Salads</i>					
Cauliflower.....	2.48	0.34	1.21	3.44	0.91
Butter kale.....	3.01	0.54	1.47	5.72	1.20
White cabbage.....	3.99	0.90	1.21	10.42	1.88
Brussels sprouts.....	4.83	0.46		6.22	1.57
Savoy cabbage.....	3.31	0.71	1.29	4.73	1.23
Red cabbage.....	1.83	0.19	1.74	4.12	1.29
Pointed head cabbage.....	1.80	0.20	1.39	2.40	0.97
Spinach.....	3.49	0.58	0.10	4.34	0.93
Rhubarb.....	2.42	0.48	0.37	3.06	0.66
Turnips (white) stem, fresh.....	2.00	0.14		1.94	1.17
String beans.....	1.73	0.17	0.66	3.97	0.88
Beans, pods only, very young.....	1.99	0.13			0.82
Bean salad.....	2.24	0.09	1.23	4.23	1.13
Princess beans.....	2.42	0.16			1.08
Lima beans, unripe.....	6.08	0.39		4.48	2.12
Green peas, very young.....	5.54	0.56		8.03	1.41
Kohlrabi, knobs.....	2.87	0.21		9.29	1.68
“ leaves and stem.....	2.03	0.45	0.38	7.80	1.55
Radishes.....	1.23	0.15	0.51	6.77	0.75
Spanish radish.....	1.92	0.11	0.88	2.91	0.75
			1.53	6.90	1.55

	Nitrogenous Matter	Fat	Sugar	N-Free Extractive Matter	Cellulose
<i>Vegetables, Salads (Continued)</i>					
Horseradish.....	2.73	0.35	15.89	11.03	2.78
Celery (<i>knob</i>).....	1.48	0.39	0.77	7.87	1.40
“ (leaf).....	4.64	0.79	1.26	1.35	1.41
Cucumber.....	1.18	0.09	0.96	4.40	0.78
Melon.....	1.00	0.32	2.13	5.16	1.09
Squash.....	1.10	0.13	1.34	2.26	1.22
Asparagus.....	1.79	0.25	0.37	12.61	1.04
Skirret.....	1.04	0.50	2.19	10.10	2.27
Carrots.....	3.52	0.14	1.24	9.02	1.82
Beets (red).....	1.37	0.03	0.54	5.65	1.05
Onion.....	2.58	0.58	2.53	1.54	1.76
Tomato.....	1.25	0.33	0.44	6.09	0.84
Leek.....	2.83	0.29	3.44	17.62	1.49
Chicory.....	1.01	0.49	1.00	3.60	0.97
Sauerkraut.....	1.00	0.20	2.19	3.55	1.80
Lettuce.....	1.41	0.31	0.76	1.82	0.73
Romaine.....	1.26	0.54			1.17
Endive.....	1.76	0.13			0.62
Prickly lettuce.....	2.09	0.41	2.73	7.45	0.57
Dandelion.....	2.81	0.69	7.13		1.96
Nettle.....	5.50	0.67			1.52

	Nitrogenous Matter	Fat	Sugar	N-Free Extractive Matter	Cellulose
<i>Vegetables, Salads (Continued)</i>					
Waybread.....	2.65	0.41	11.19		2.09
Portulaca.....	2.24	0.40	2.16		1.03
White goosefoot (<i>Chenopod</i>).....	3.94	0.76	8.93		3.82
Potato.....	2.08	0.15	0.28	20.73	0.69
Turnip.....	1.08	0.22	5.89		1.13
Cheeril.....	3.89	0.32	1.60	26.23	0.94
Carrot, small variety.....	1.07	0.21	1.58	6.59	0.98
Parsnip.....	1.37	0.44	2.06	10.50	1.42
Sugar beet.....	2.87	0.34	4.50	15.19	2.11
Batata.....	1.00	0.20	1.74	23.31	1.03
Sweet potato.....	1.78	0.14	14.04		0.64
Egg plant.....	0.88	0.10	3.61		1.28
Mushroom.....	3.74	0.15	0.75	2.76	0.84
Truffle.....	8.60	0.62	8.10		7.57
Morel.....	3.48	0.24	0.10	4.57	0.67
Chantarelle.....	3.92	0.52	1.17		1.64
Boletus.....	3.61	0.17	3.72		0.57
“ other varieties.....	1.59	0.26	2.53	2.87	0.92
Clavaria.....	2.44	0.21	5.99		0.69
Ogaricus.....	3.04	0.35	5.90		1.04

	Nitrogenous Matter	Acid	N-Free Extractive Matter = Pectin*)	Sugar Total	Levulose Alone	Cellulose (Kernels,pits)
<i>Fresh Fruits</i>						
<i>Cranberry</i>	0.12	2.34	—	1.53	1.00	6.27
<i>Green Gage</i>	0.41	0.91	11.46	3.16	2.00	3.39
<i>Plums</i>	0.40	1.50	4.68	3.56	2.30	4.34
<i>Raspberry</i>	0.40	1.42	1.44	3.86	2.60	6.66
<i>Yellow plum</i>	0.38	0.53	10.07	3.97	2.65	4.99
<i>Blackberry</i>	0.51	0.19	1.44	4.44	2.96	5.21
<i>Peach</i>	0.65	0.92	7.17	4.48	3.00	6.06
<i>Orange</i>	0.73	2.44	0.95	4.58	3.00	1.78
<i>Apricot</i>	0.49	1.16	6.35	4.69	3.15	5.27
<i>Huckleberry</i>	0.78	1.66	0.87	5.02	3.34	12.29
<i>Prunes</i>	0.78	0.85	4.92	6.15	4.00	5.41
<i>Strawberry</i>	1.07	0.93	0.48	6.28	4.00	2.32
<i>Currant</i>	0.51	2.15	0.90	6.38	4.25	4.57
<i>Gooseberry</i>	0.74	1.42	1.40	7.03	5.00	3.52
<i>Apple</i>	0.36	0.82	5.81	7.22	4.80	1.51
<i>Pear</i>	0.36	0.20	3.54	8.26	5.50	4.30
<i>Mulberry</i>	0.36	1.86	2.03	9.19	6.13	0.91
<i>Cherry</i>	0.67	0.91	1.76	10.24	7.00	6.07
<i>Tamarind</i>	—	14.65	10.90	12.50	—	32.10
<i>Grape</i>	0.59	0.79	1.96	14.36	—	3.60
<i>Dried Fruits</i>						
<i>Apples</i>	1.28	3.60	16.96	42.83	28.00	4.99
<i>Pears</i>	2.07	0.84	20.67	29.48	20.00	6.86

*Pectin = a vegetable body combined with lime which is not converted into sugar.

	Nitrogenous Matter	Acid	N-Free Extractive Matter = Pectin*)	Sugar Total	Levulose Alone	Cellulose (Kernels, pits)
<i>Dried Fruits (Continued)</i>						
<i>Plum jam</i>	2,25	2,75	17,91	44,41	30,00	1,52
<i>Cherry jam</i>	2,07	0,30	14,29	31,22	28,00	0,61
<i>Raisins</i>	2,42	0,59	7,48	51,56	40,00	1,72
<i>Sultanas</i>	—	1,48	1,67	61,88	34,00	—
<i>Currants</i>	—	—	1,48	61,88	—	—
<i>Figs</i>	4,01	—	—	49,79	—	15,00
<i>Dates</i>	—	—	—	61,40	—	12,30
<i>Halva</i>	2,99	11,00	36,18	19,44	16,16	9,87
<i>Banana</i>	1,87	0,63	23,05†	—	—	0,29
<i>Sugar in Pure Fruit Juices</i>						
<i>Pear</i>				11,5	0,13	—
<i>Apple</i>				7,9	0,51	—
.....				12,7	0,72	—
<i>Currants, white</i>				7,84	2,39	—
<i>red</i>				6,89	2,71	—
<i>Cherry</i>				13,82	0,88	0,68
<i>Mahaleb cherry</i>				10,06	2,23	—

*Pectin = a vegetable body combined with lime which is not converted into sugar.

†Cane sugar 15,90%; Invert sugar 8,50%; Starch 0,60%

				Levulose per cent	Acid per cent	Cane Sugar per cent
<i>Sugar in Pure Fruit Juices (Continued)</i>						
Wild strawberry.....				4.15	1.23	0.17
Cultivated strawberry.....				6.89	1.03	1.37
Huckleberry.....				7.76	1.20	—
Raspberry.....				6.97	1.94	—
Gooseberry.....				7.25	1.55	—
Lemon juice.....				—	7.00	—

					Nitrogenous Matter	Fat	N-Free Extractive Matter	Cellulose
<i>Nuts</i>								
Butternut.....					28.00	61.1	3.5	4.15
Walnut.....					16.37	62.86	7.89	6.17
Filbert.....					15.62	66.47	9.03	3.28
Peanut.....					28.16	46.37	1.85	13.87
Cocanut.....					5.49	35.93	8.06	2.91
“ milk.....					0.46	0.07	6.78	—
Almond.....					24.18	53.68	7.23	6.56
Brazil nut.....					15.30	65.00	7.39	3.9
Chestnut, fresh.....					6.2	5.5	42.2	13.5
“ dried.....					10.7	7.0	74.1	—

Wines and Other Alcoholic Beverages

	Alcohol	Extract	Tartaric Acid	Sugar	Glycerin	Tannin and Coloring Matter
<i>White Wines</i>						
Moselle.....	7,99	2,24	0,79		0,72	
Winninger Mount, 1876.....	8,22	1,99	0,64		0,66	
Bockstein, 1876.....	8,36	2,53	0,78	0,027		
Saarburg, 1881.....	7,87	2,76	1,05	0,02		
Scharzhofberg, 1857.....	14,2	2,00	0,56	0,15		
Rhine wine.....	8,00	2,60	0,81		0,85	
Rehbach, 1875.....	9,35	2,25	0,80		1,08	
Steinberg, 1882.....	5,30	2,88	1,30		0,49	
Hattenheim, 1882.....	6,86	3,37	1,03		0,77	
Geisenheim, 1881.....	8,60	3,21	1,25	0,15	0,92	
Rhenisch-Hessian.....	9,91	2,52	0,56		1,02	
Palatinate.....	8,11	2,43	0,67		1,12	
Deidesheim, 1878.....	8,91	2,28	0,70		1,18	
Forster Ziegler, 1876.....	9,88	2,48	0,54	0,11	1,28	
Frankisch wines.....	7,75	2,31	0,74	0,18	0,90	
Stein, 1876.....	9,00	2,22	0,68		0,92	
Leisten, 1876.....	10,15	2,41	0,80		0,86	
Spiegelberg, 1878.....	8,24	2,57	0,60		1,18	
Tauber wine.....	7,71	2,38	0,70		0,91	
Baden wine.....	7,42	2,35	0,39		0,61	
Markgräfler Kastelberg, 1868.....	8,83	1,80	0,59			
“ “.....	8,83	2,02	0,86			
Württemberg.....	6,10	2,27	0,95		0,57	

	Alcohol	Extract	Tartaric Acid	Sugar	Glycerin	Tannin and Coloring Matter
<i>White Wines (Continued)</i>						
Alsatian wines	8,07	1,73	0,51	0,09		
Alsace-Lorraine	6,41	2,02	0,71		0,55	
French wine	10,31	3,03	0,66		0,97	
Chat. Filhot, 1877	12,49	3,54	0,75		1,03	
Hautes Sauternes	10,27	3,19	0,65		0,97	
Tyrolese	8,84	1,87	0,59		0,65	
Lower Austrian	7,93	2,13	0,67		0,68	
Styrian	8,44	3,14	0,78		0,71	
Hungarian	8,00	2,33	0,69		0,77	
<i>Red Wines</i>						
Ahr wine	8,02	2,58	0,48			0,20
Walportshcim, 1868	10,63	2,65	0,41	0,16		0,23
Rheingau	10,08	3,04	0,52			0,16
Assmanshausen, 1868	10,41	2,86	0,47			0,25
Rhenisch-Hessian	7,70	3,01	0,58			0,15
Palatinate	9,39	2,94	0,49		1,29	
Baden	7,76	2,16	0,95	0,09		
Württemberg	5,92	2,64	1,14		0,46	
Alsace	8,88	2,27	0,56		0,72	0,16
Lorraine	8,08	2,89	0,46		0,50	0,29
Swiss	8,00	2,31	0,79		0,61	0,18
Tyrolese	9,08	1,87	0,62		0,65	0,11
Lower Austrian	8,49	2,54	0,62		0,81	0,18
French Bordeaux	7,80	2,56	0,57	0,30	0,73	

	Alcohol	Extract	Malic Acid	Sugar	Glycerin	Tannin and Coloring Matter
<i>Fruit Wines</i>						
Apple cider.....	5.80	2.56	0.50	0.45	0.78	
Pear ".....	3.65	3.68	0.93	0.14	0.37	
Apple " Swiss.....	4.80	2.53	0.54	0.37	—	
Pear ".....	4.01	3.17	0.48	0.70	—	
Current wine.....	11.14	12.69	1.06	9.86	0.57	
".....	11.31	2.28	1.19	0.00	0.48	
Gooseberry wine.....	11.22	13.08	0.75	9.80	0.77	
".....	21.31	3.93	0.95	1.89	0.46	
	Alcohol	Extract	Tartaric Acid	Sugar	Glycerin	Tannin and Coloring Matter
<i>Sparkling Wines</i>						
French Brand 1.....	10.20	19.75	0.60	17.52	1.13	
" " 2.....	9.50	20.24	0.70	18.50	0.97	
" " 3.....	9.03	13.37	0.58	11.54	?	
" 2nd quality.....	8.14	8.13	0.55	6.39	0.28	
German Brand 1.....	9.44	10.47	0.57	8.70	?	
" " 2.....	9.70	13.23	0.66	11.25	0.74	
" " 3 (red).....	10.50	10.76	0.46	8.64	0.81	
" " 4.....	9.13	4.99	0.58	2.61*	0.81	
Sparkling cider.....	6.21	10.00	0.63	7.44	—	0.06

* Levulose 1.28. Dextrose 1.33.

	Alcohol	Extract	Tartaric Acid	Sugar	Glycerin	Tannin and Coloring Matter
<i>Sweet Wines</i>						
<i>Tokay, dry</i>	12.05	3.26	0.68	0.63	1.04	
“ <i>from picked grapes</i>	9.44	23.63	0.57	19.44	0.62	
<i>Sherry</i>	17.45	3.98	0.45	2.12	0.52	
<i>Madeira</i>	15.40	5.52	0.43	3.23	0.74	
<i>Marsala</i>	15.85	5.27	0.49	3.53	0.51	
<i>Port wine</i>	16.69	8.05	0.40	5.82	0.43	

	Alcohol	Extract	Sugar
<i>Liquors</i>			
<i>Rye whiskey</i>	45.1	—	
<i>Cherry brandy</i>	43.9	0.05	
<i>Prune brandy</i>	28.7	0.06	
<i>Grape husks</i>	42.5	0.03	
<i>Whiskey</i>	52.2	0.04	
<i>Cognac</i>	43.9	0.38	
“ <i>from grapes</i>	69.5	0.65	
<i>Absinthe</i>	58.9	0.77	
<i>Rum</i>	61.4	1.97	
<i>Arrack</i>	52.5	0.08	
<i>Gin</i>	47.8	1.75	1.40

	Alcohol	Extract	Carbonic Acid	Sugar	Gum Dextrin	Proteids
<i>Beer</i>						
<i>Winter brew, light</i>	3,46	5,49	0,19	0,95	3,11	0,81
<i>Lager beer, heavy</i>	3,95	5,78	0,19	0,68	4,70	0,44
<i>Export beer</i>	4,31	6,48	0,21	1,06	—	0,44
<i>Ale</i>	4,89	6,03	0,21	0,84	—	0,53
<i>Porter</i>	5,35	7,56	0,15	1,34	—	0,69
<i>Mumme Braunschweig</i>	2,96	5,2,29	0,12	—	—	—
<i>Pilsener, exportation</i>	4,60	4,42	—	?	—	—
“.....	3,47	4,97	—	?	—	0,37
<i>Weissbier</i>	2,73	5,34	0,29	1,62	2,42	0,58

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